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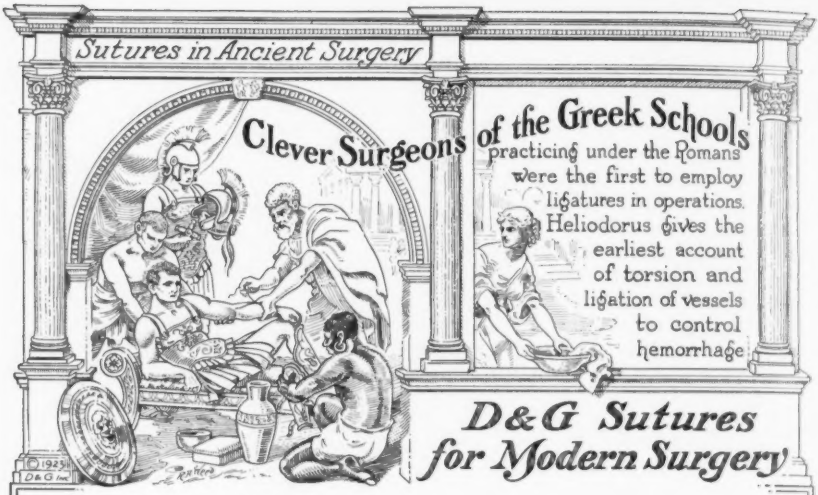
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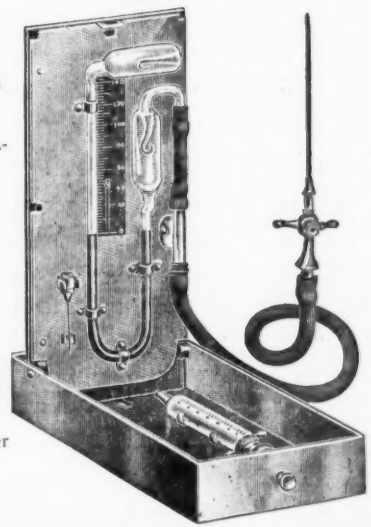

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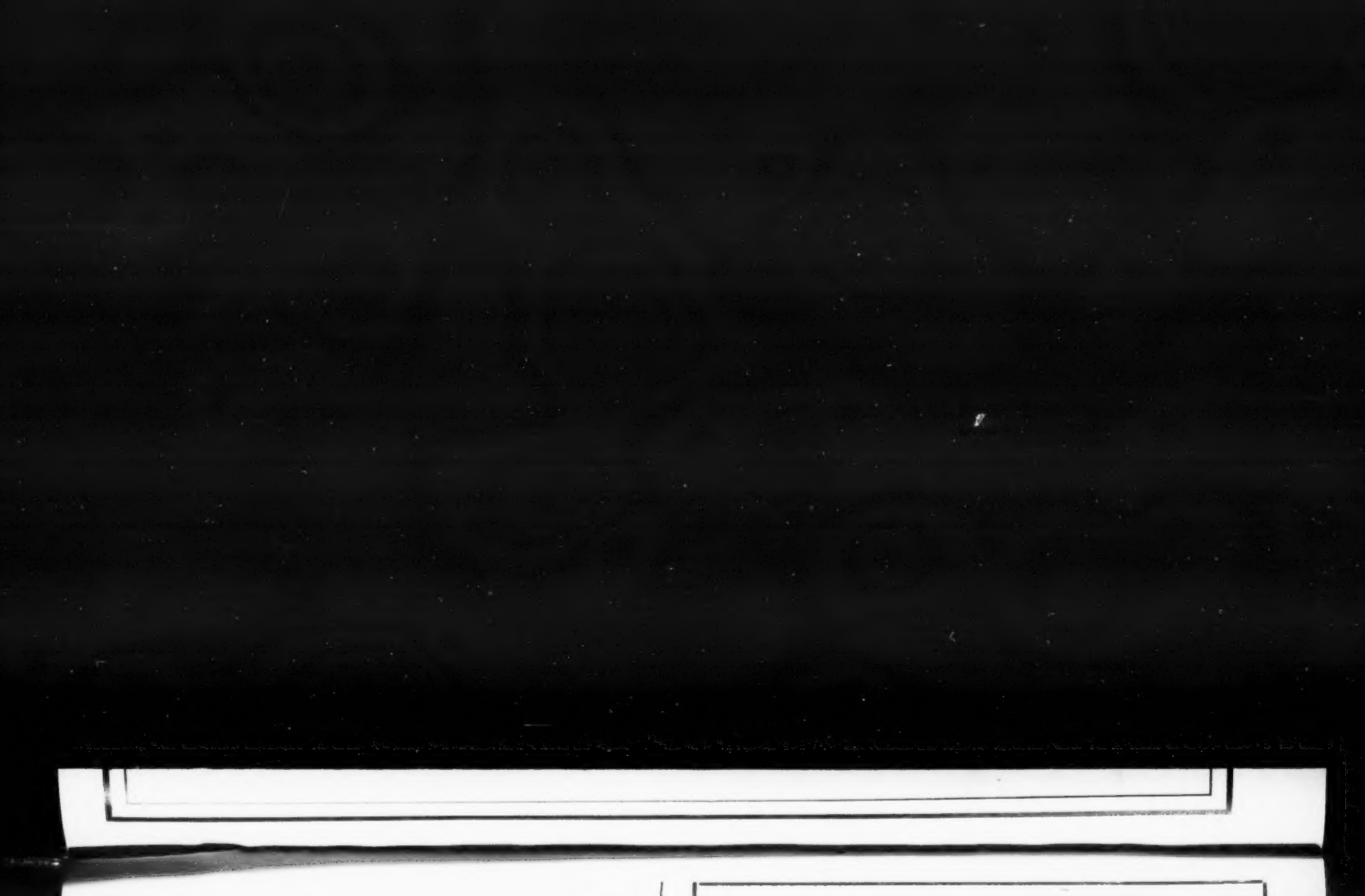

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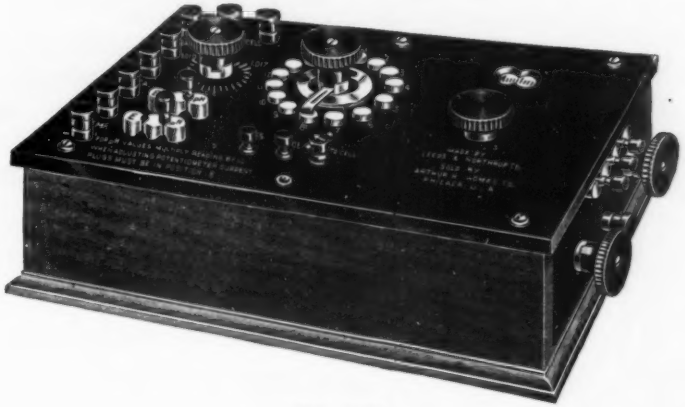

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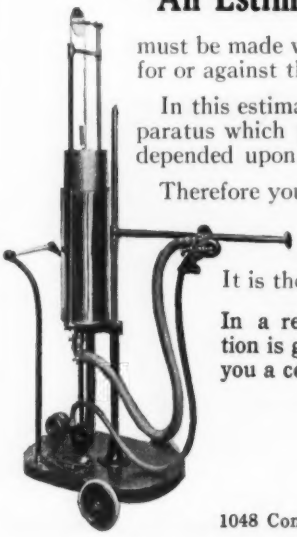

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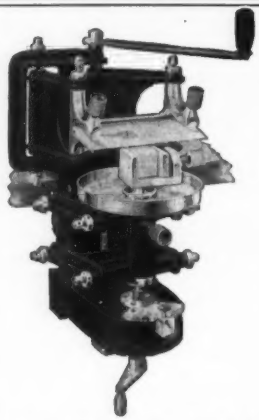

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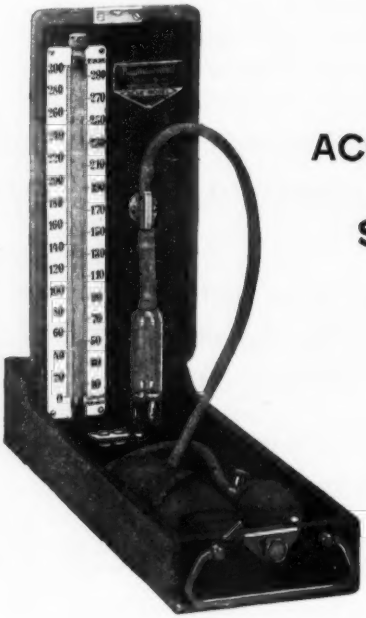

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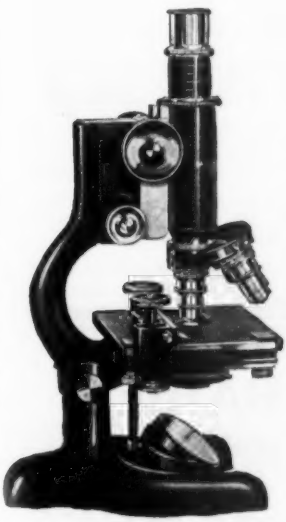

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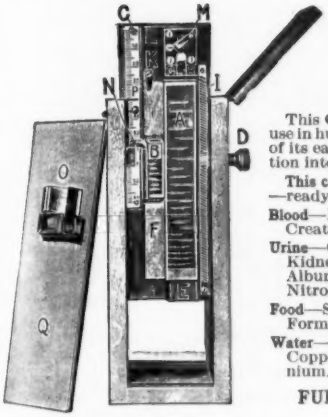


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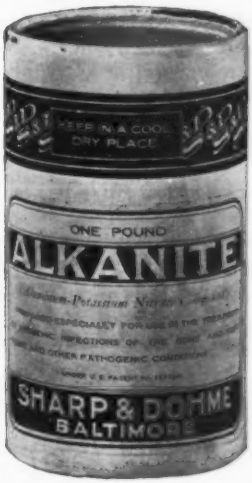

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## OF THE MEDICAL SCIENCES

SEPTEMBER, 1923

## ORIGINAL ARTICLES.

### THE PROGNOSIS OF SYPHILIS.

By John A. Fordyce, M.D.

NEW YORK.

Because of the extensive and intensive educational propaganda regarding the prevalence and seriousness of venereal diseases a large percentage of the educated lay public has a superficial knowledge of their nature and results. This information is sometimes misleading and conveys impressions which tend rather to exaggerate than to properly measure their importance. After a patient with a syphilitic infection recovers from the first mental shock of the physician's diagnosis his next reaction is to ascertain the possibility of cure, the duration of treatment and the chances of developing certain sequelæ which he fears may follow. Will he ever be fit for matrimony and capable of begetting healthy children? How long is the disease contagious? These among other questions, confront all physicians who have to do with this class of cases.

Is the medical adviser who assumes the responsibility of treating and predicting the outcome of a case of syphilis sufficiently familiar with the course of the disease and with the effects of the specific remedies to be able to give an intelligent answer to these questions? Can he assure the anxious patient that a cure will follow treatment over a given period of time and that the most feared late results of the disease will not occur?

It is unfortunate that the majority of practitioners see only that phase of the disease which occurs in their particular work. Few have the opportunity to follow that patient from the initial infection through the varied possible clinical manifestations. Their

prognostic views are influenced by the phase of the disease they observe and the manner in which it responds to treatment. The visible accidents are treated until they disappear but the infection as a whole is seldom treated or followed by any sustained effort. It is only by piecing together scattered observations by clinicians, pathologists and serologists that we can obtain an imperfect conception of the prevalence and possibilities of this great infection.

An examination of the vital statistics when properly compiled supplies accurate information as to the cause of death in all diseases excepting the venereal group. It is comparatively easy to determine the average mortality from typhoid fever and to predict with a fair degree of assurance the outcome of a given case. Syphilis seldom or never appears in mortality statistics as the direct or indirect cause of death. It is not written on death certificates. The relation of cause and effect is lost sight of because of the long duration of the infection and the slowly progressive nature of the tissue degenerations which arise from it. Definite names which carry more respectability, such as myocarditis, aneurysm, cerebral hemorrhage, the renal complications of tabes are given as the direct cause of death, and the relatives and friends are comforted and deceived.

We have, therefore, at the present time no correct estimate of the incidence of syphilis owing to the large number of unsuspected and undiagnosed cases as well as wilfully concealed diagnoses. The statistics available give only the approximate prevalence as based on positive Wassermann reactions obtained in the army and navy, in general hospitals, institutions for the insane and in certain industries where the test is required, as among those who handle food. In hospitals positive Wassermann reactions have been obtained in 20 to 30 per cent of admissions; in insane asylums in 20 per cent. The percentage for our entire population is variously given as 5 to 20. Insurance statistics show that the mortality of the syphilitic is 33.3 per cent above expectations. Autopsy records, too, reveal a wide discrepancy according to the pathological criteria employed in the studies. Deaths from syphilis occur in individuals forty to sixty years of age as the result of myocardial, vascular, renal and hepatic disease not clinically recognized as syphilis. In 4880 autopsy records at Bellevue Hospital, Symmers found syphilis in 314 cases or 6.5 per cent. In a similar study by Warthin at Ann Arbor, during a ten-year period, the infection was found present in 300 cases or 40 per cent of the number investigated. Symmers' diagnosis was based on gross syphilitic changes such as aortitis, aneurysm, chronic interstitial orchitis and disease of the central nervous system, liver, bones, respiratory tract, and so on. Warthin's criteria were small focal lesions found in the various organs in addition to easily recognized gross changes.

It would be unscientific and illogical to prognosticate the future

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of a syphilitic patient seen at an early stage of the infection and treated by modern specific drugs from the viewpoint of the pathologist or from the possible end-results of undiagnosed and inadequately treated cases. Fortunately, all syphilitics do not die of the infection. Cases aborted or cured by early treatment do not often favor us by dying of some intercurrent malady. We are, therefore, denied the absolute proof of cure insisted on by the pathologist and are obliged to rely on the revelations of the Wassermann reaction properly interpreted and controlled by the clinician, the occurrence of second infections, and the prolonged absence of clinical symptoms.

It would be a superhuman task to convince certain pathologists who claim that all syphilitics who come to autopsy with or without a positive Wassermann reaction are ever cured. On the other hand, it would be a task fully as difficult to convince an experienced clinician that syphilis treated at the opportune time and in the proper manner cannot be cured. Fortunately, the victim of the disease is not advised by the pathologist and is spared a lifetime of misery and fear by the sane knowledge of the clinician. He is cured, marries and has healthy progeny if fortunate enough to fall in the hands of a practitioner equipped with modern tools and the knowledge of how they should be used.

In the pre-Wassermann days the disease was said to be cured by so many years of treatment by the favorite pill or mixture of the doctor and the absence of visible signs of the disease. Syphilis was considered by many as less serious than gonorrhea. Jonathan Hutchinson repeatedly states in his numerous contributions to the clinical side of syphilis that the vast majority of those who have passed through an attack get well with or without treatment and remain well during the rest of their lives. The optimism of Hutchinson while concurred in by some of his colleagues was as vigorously denied by others who were more inclined to state that "once syphilis always syphilis." These opinions were largely due to impressions based on a more or less large clinical experience without a clear conception of the possibilities of the infection.

The prognosis of syphilis based on a larger grasp of the problem and the modern criteria of cure is on an entirely different basis than in the pre-Wassermann era. Then the absence of clinical symptoms, especially the external manifestations, was taken to indicate a cure of the infection. Immunity to new infection was considered in the same light as immunity to the acute exanthemata. The occurrence of second infections was rare and even disputed. Now numerous cases are reported in literature and are seen in every large clinic devoted to syphilis.

The physician who assumes the responsibility of treating and predicting the outcome of a case of recent syphilis or a case of syphilis in any stage, is handicapped by a lack of knowledge as to

all its possibilities. If he does not grasp the relation of early involvement of the central nervous system to its late degenerations how can he predict to an individual that he will or will not at some future time die from cerebral hemorrhage or become the victim of paresis or tabes? To the internist come the cases of aortic disease, myocarditis and aneurysm due to slowly progressive changes over years. The syphilographer should have enough knowledge of internal medicine to be able to visualize the future of his patient along these lines. He should in a word know all the tissue reactions evoked by the invasion of the treponemata and to what degree they can be influenced by our therapeutic agents. A knowledge of the pathological possibilities of the infection is one of the factors on which we base our prognosis.

**The Wassermann Reaction as a Guide in Prognosis.** In the vast majority of cases of syphilis a close correlation exists between the signs and symptoms of the disease and the complement-fixation test. Experience has taught us, however, to modify some of the views originally held. We should not, however, emphasize the exceptional and incidental phases of the reaction and forget the rule which generally holds good.

The report of cases of neurosyphilis in which negative findings were present in the spinal fluid with positive signs or symptoms of the central nervous disease, and conversely, cases with positive findings in the fluid and no symptoms is of much value in calling attention to exceptional conditions. Such reports are, however, misleading if the impression is conveyed that spinal fluid findings are valueless in the diagnosis and prognosis of neurosyphilis. Paresis in practically 100 per cent of cases has a definite formula in the fluid. The absence of the paretic gold-sol curve enables us to exclude paresis in our prognosis. The majority of active progressive tabetics have definite fluid changes which are of prognostic value and an accurate guide to treatment.

Some years ago a repeated negative reaction in the blood was looked upon as an accurate criterion of cure. We now know that many such patients have a positive reaction in the spinal fluid, while the blood remains persistently negative. Furthermore, a weakly positive reaction in a patient with a definite history of syphilis is an indication for further treatment. A reaction of this kind reported from the laboratory is usually considered a negligible one. Before this interpretation is placed upon it the history of the patient, together with the amount of treatment he has received, should be carefully considered. In expressing an opinion, therefore, regarding the question of cure, or the absence of a syphilitic infection, the Wassermann reaction of the blood, as well as of the spinal fluid, should be carefully correlated with the previous history of the patient. I have often been aided in my interpretation of a Wassermann reaction by opinions from my colleagues as to the

clinical findings which are present. In several cases an eyeground examination has shown the existence of vascular disease with degenerative changes in the presence of a negative blood. In some cases the vascular degeneration is a progressive one and may be met with in the early stages of syphilis. It would, therefore, be fatal to such patients to state that the syphilitic infection has been cured. It is in cases like these, and similar ones with localized lesions, that we obtain a negative reaction in the presence of active disease. Some of these patients two or three years later may show evidence of hypertension and it is a question whether all syphilitics should not be under observation from time to time for a cardiac and ophthalmological examination and whether our intensive arsphenamine-mercury treatment should not be followed up by iodides.

The possibilities of error in the interpretation of the Wassermann reaction are more numerous on the negative than on the positive side and we might briefly formulate them as follows: The blood is negative in about 6 per cent of late active cutaneous syphilis and sometimes in early malignant syphilis; in a certain percentage of cases with cardiovascular syphilis; in certain localized progressive vascular affections of the eye; occasionally in congenital syphilis although the infant has clinical signs of the disease. The blood usually becomes strongly positive after treatment.

In the following types of neurosyphilis the spinal fluid is often negative and the blood may be negative or positive; in vascular neurosyphilis; in abortive tabes and the late degenerative stages of the disease and sometimes in the terminal stages of paresis. Epilepsy, cerebral gumma and syphilitic psychoses are also usually accompanied by a negative Wassermann reaction in the fluid.

It is well to keep in mind that the Wassermann reaction has been tested in hundreds of thousands of cases and has stood up better than any other laboratory procedure. It is one of the symptoms of syphilis and like others may occasionally be absent. Not infrequently it is the only symptom of the infection when the lesion producing it cannot be uncovered. It requires confirmation by more than one laboratory together with a review of the patient's general history and a careful consideration of the theoretical possibility that other conditions might cause it.

In summarizing one can say with a certain degree of assurance that a persistent positive reaction controlled in several laboratories means active treponemata in some part of the body and is an indication for treatment. Autopsy findings in cases with a history of syphilis who have died from some intercurrent affection show that in over 90 per cent the aorta is the site of a lesion and that in over 40 per cent there is involvement of the viscera.

A persistent negative reaction in the blood and spinal fluid after intensive treatment is an indication of cure in the great majority of cases. The cured cases of syphilis do not go to autopsy

and this statement is not susceptible of pathological proof, but the subsequent histories of these patients with their marital history and healthy children support this view. The exceptional cases on the negative side which have been referred to cannot take away from the value of a negative Wassermann reaction and neither can they be accepted as proof that syphilis is an incurable disease.

Modern treatment is without doubt more effective in curing the disease than before the introduction of the arsenic compounds and the more systematic and better methods of employing mercurials. It is generally conceded that treatment begun at the earliest possible time before the development of a positive Wassermann reaction offers the best hope of aborting the infection. Opportunities for employing abortive treatment are often missed by failure to diagnose extragenital chancres, atypical or insignificant looking lesions and so-called mixed sores. In women the primary sore is seldom seen and here, as well as in the infections which take place at the time of conception and during pregnancy, the favorable time for abortive treatment is long passed before specific remedies can be employed. In the large group of cases of congenital and antenatal syphilis the ignored infection overwhelms the victim before therapeutic help is possible or of much avail. It has been estimated that in 70 per cent of all cases of syphilis treatment is not begun until the stage of generalization is well advanced. The ideal time for abortive treatment is often missed by conditions beyond our control. In cases where the primary sore is evident, the lack of clinical knowledge and inability to properly employ the dark-field microscope leads often to loss of valuable time. Failure to demonstrate the treponemata in the chancre may be rewarded by success in the fluid aspirated from the communicating lymph node. More careful and detailed instruction is necessary in our medical schools in the diagnosis of the objective appearance of the primary sore as well as in the employment of the dark-field microscope. The average time for the Wassermann reaction to appear in the blood should always be kept in mind as many mistaken diagnoses are made by blindly following the laboratory reports without proper correlation with the clinical picture. Clinical experience has amply demonstrated that abortive treatment is successful at this stage of the infection even though animal experiments have shown a diffusion of the organism beyond the site of the primary sore and its lymphatic connection. We now know that immunity to a new infection in syphilis means that the body still harbors the parasite and in this respect differs from the bacterial diseases which confer a life-long immunity and in which the causal agent disappears. A reinfection in syphilis assumes the complete disappearance of the treponemata and their protective power against a new invasion. Definite proof as to the curability of syphilis is therefore supplied by cases of reinfection which are becoming more and more numerous

as we are becoming more expert in diagnosis and in handling our specific drugs. The following cases which among others have come under the writer's observation are cited as illustrations:

P. P., a male, aged thirty-one years, had a penile chancre in November, 1918. He was admitted to the clinic with mucous patches in January, 1919. The Wassermann was 4 plus. The treatment consisted of 25 injections of arsphenamine and 13 of mercury salicylate. The Wassermann on three different occasions was negative, the last in September, 1919. He then disappeared and was not seen again until September 15, 1920, when he returned with an indurated sore in the sulcus of the penis of two weeks' duration. The dark field showed many treponemata. Wassermann 4 plus. Exposure was admitted.

D. S., a male, aged twenty-five years, came to the clinic in August, 1920, with a chancre of the penis and inguinal adenopathy. Dark-field examination, positive; Wassermann reaction, negative; no skin or mucous membrane lesions. Treatment was begun immediately. Blood a week later was reported strongly positive. He received 16 injections of arsphenamine and 22 of mercury salicylate in two courses. A spinal fluid examination made after the first course was negative in all phases. The blood, which was also negative at this time, was tested at regular intervals and continued negative, the last examination being made on January 5, 1922.

On February 15, 1922, the patient consulted us for a superficial clean-cut lesion on the shaft of the penis opposite to his previous ulceration and of four or five days' duration. He admitted exposure four weeks previously. The dark field showed numerous treponemata. Wassermann, negative; no glandular enlargement; no cutaneous or mucous membrane lesions. To determine whether this was a true reinfection, saline dressings only were ordered and he was again examined on February 21, when the lymph nodes on the side of the lesion were found to be distinctly palpable and fluid aspirated from one of them contained many treponemata. The Wassermann continued negative until February 28, when it was reported +. The epitrochlear glands were now slightly enlarged. Saline dressings were continued. On March 6 the blood was ++++ and on March 14 a typical secondary maculopapular eruption made its appearance on the trunk.

It is neither expedient nor desirable to subject our supposedly cured cases to the rigid test of cure, namely, a new infection, so we are obliged to rely on a persistent negative Wassermann reaction in the blood and spinal fluid, a long absence of signs and symptoms and the revelations of matrimony with its resulting progeny.

Every practitioner who has followed the life history of infected individuals knows that many die in advanced life from diseases other than syphilis even with a persistent positive Wassermann reaction. I am familiar with a man almost eighty years of age with a positive reaction due to a syphilitic infection over fifty years ago. He is in excellent health and appears much younger than his years. He has no subjective or objective evidence of his old infection. A condition of perfect parasitism has become established between the organism and the host so that they now dwell together in peace and harmony. Time and antibody formation in certain cases gradually weaken the virus and fibrosis obliterates the activity of focal lesions in organs with a large margin of safety.

Marriages and healthy children are met with in individuals with persistent positive Wassermann reactions. A focal lesion in the tongue existed for years in a man who came under my care, the result of an infection years before he married. His wife gave birth to several healthy children and all were negative to the Wassermann reaction. We frequently see men and women with late syphilis of the skin the parents of healthy children. It is not always good judgment to withhold our consent to matrimony in individuals years after infection whose reaction persists after prolonged treatment. The world contains many robust individuals born of parents previously syphilitic. The late lesions of syphilis are often localized. Their carriers neither convey nor transmit the disease.

Syphilis is a disease of so many contradictions and exceptions that the foregoing statement must be accepted with reservations. They may be the exceptions which prove the rule, but they should dissipate a part of the pessimism as to possibility of cure which depresses the victim and impairs the physician's will to cure. Clinical medicine is a vast workshop in which experimentation, observation and interpretation yield results as important to our art as the findings in the purely experimental laboratory. Each must control the work of the other but neither must assume an attitude of intolerence. The problems of syphilis appeal to the clinician in every field of medicine and equally to the laboratory worker. With a proved etiology, accurate methods of diagnosis, specific drugs and a fairly accurate measure of their effects the practical application of this knowledge should lead to a large percentage of cures.

It is safe to state that the problem of cure is approached by the majority of practitioners with little exact knowledge of the pathology of the disease and with less knowledge as to the employment of the specific drugs. The early stage of neurosyphilis is often entirely overlooked and not considered in its relation to the late degenerations; vascular and eye lesions are ignored until irreparable damage is done. A cure is supposed to follow the administration

of a definite number of intravenous injections of arsphenamine with solutions improperly prepared and crudely given. If cure does not result the patient is at fault and the doctor holds himself blameless. Under these conditions complete clinical and serological cures are the exception rather than the rule and the defensive forces of the body weakened by insufficient treatment permit foci to develop in the central nervous system and elsewhere. Insufficient and improperly employed therapeutics is often more harmful than helpful and may precipitate in an early period lesions usually seen late in the infection. If we admit the increased incidence of neurosyphilis since the arsphenamine era its explanation is to be found in insufficient early treatment and in the routine administration of so many doses of arsenic and so much mercury regardless of the individual's requirements.

Observations and analysis of cases over a period of years that were properly treated has failed to convince me that neurosyphilis has been rendered more frequent by the modern method of treatment. In the early days of arsphenamine administration when a few doses of the drug were considered the maximum requirements the incidence of neurosyphilis may have been more frequent. British observers have remarked an increase in neurosyphilis among cases treated in the army where a fixed number of injections were given to cure the existing lesions and return the man to active duty at the earliest possible time; a symptomatic cure only was attempted. Since we have insisted on an examination of the spinal fluid before a final pronouncement of cure and proper treatment of a pathological condition, if present, we have had no case of early syphilis return to us with the so-called neuro-recidives. In other words, if the central nervous system is not involved early in the infection, it is highly improbable that it will be implicated later. If it is found to be involved in the early period during active treatment and fails to yield to additional treatment along the usual lines it will yield to intraspinal injections. The lesson to be learned from the discussion as to the increased incidence of neurosyphilis and the weakening of the antibody defence by modern treatment is to give the remedies in suitable doses at proper intervals and over a sufficiently long time to destroy all the invading organisms. Too long rest periods between courses of treatment permit the surviving treponemata to begin anew their activities. Neurosyphilis may show an increased incidence in cases improperly treated. It has, however, always existed and its frequency has been brought to the foreground by our modern methods of investigation and the attention which has been focussed on it.

We can speak with a certain amount of assurance as to the prognosis of cases of early syphilis if coöperation is brought about between the physician familiar with the possibilities of the disease,

modern therapeutic methods and a patient with ordinary intelligence. The future of such patients depends on careful supervision over a period of years. A control of treatment by the Wassermann reaction in the blood as well as the knowledge gained by lumbar puncture are imperative.

Attention focussed entirely on the reaction of the blood with neglect of the spinal fluid has in many cases led to disastrous consequences and irreparable conditions. A patient discharged after adequate treatment with a conservative opinion as to the cure with a persistent negative blood and negative fluid has up to this time never returned with clinical or serological evidence of neurosyphilis. On the contrary, we have many records of patients discharged with a negative blood only who have come back to us after months or years with definite evidence of nervous system involvement. The prognosis of early syphilis is therefore intimately related to the cyto-biological changes in the fluid as well as the Wassermann reaction in the blood. The development of persistent hypertension in these cases after what is considered adequate treatment is another condition which demands further study. It is an occasional sequence of the infection in young adults where the usual factors cannot be invoked as a cause and may be looked upon rather as a coincidence than the result of the infection or treatment employed. Progressive low grade reactions may continue in the heart and arteries with a negative Wassermann. Possible toxic effects on the bloodvessels by arsenic should be kept in mind. It may be advisable in these cases to ignore the reaction and to follow up the usual treatment with the iodides combined with mercury in moderate doses. We are, as the result of experience, adding something to the credit side of our diagnostic and therapeutic methods and thus increasing the patient's chances of cure.

We are yet far from a standardized and ideal formula of treatment which insures the patient's future and eliminates the possible harmful effects of the drugs. In this connection a reference to the occurrence of jaundice during and after arsphenamine treatment may not be inopportune. In our endeavor to sterilize the patient rapidly the tendency is to increase the number of arsphenamine injections in our so-called course of treatment and to give additional courses. If we keep in mind the fact that after each dose of the drug a certain percentage is stored in the liver and with frequent repetitions a toxic amount may be retained, it is not difficult to grasp the relationship of the drug to the jaundice and the degenerative changes in the liver.

In my own work jaundice has appeared from a few days or weeks to three and a half months after the last dose of arsphenamine and not infrequently in groups. We have seen it after all the arsphenamine preparations, including silver arsphenamine.

Its development can sometimes be foreseen by the occurrence of certain symptoms such as severe pains in the arms and legs, lassitude and loss of appetite several weeks before the onset of the jaundice.

The incidence of other by-effects of our drugs, such as types of dermatitis, purpura, severe anemia and encephalitis hemorrhagica, while rare must also be considered in our general scheme of prognosis. While millions of doses of arsenic preparations have been administered with no untoward results, many deaths from arsphenamine have been unrecorded and too little emphasis placed on its toxic and by-effects. More technical knowledge and skill are required in our modern treatment as the possibilities of accidents are greatly increased.

The prognosis of late syphilis is a vastly more complicated problem than in early syphilis. It is concerned with phases of the disease in which damage may have resulted in organs essential to life and in individuals with the degenerations incident to age. Brain hemorrhages, thrombosis and aneurysms occur in advanced arterial disease where the specific factor is reinforced with the mechanical factor of the circulation. Changes in the heart muscle resulting in fibrosis and in the coronary arteries causing deficient blood supply to the heart may be modified but not cured by our specific drugs. Great harm may follow their indiscriminate and careless employment. A possible Herxheimer reaction in lesions involving important organs may cause alarming symptoms and even death. Where the possibility can be foreseen careful preliminary treatment must be employed. The internist and the syphilographer must work hand in hand in planning the best therapeutic procedure in cardiovascular and visceral syphilis as only in this way can possible accidents be prevented and the interest of the patient best conserved.

When the stigmata of paresis appear a deep and disseminated encephalitis is present from long continued and progressive changes in the small vessels of the brain. The brain cells are atrophic as a result of the diminished blood supply and the possible toxic effects of the treponemata. We can only offer a hopeless prognosis in spite of all the procedures that have been advocated and employed. In certain stages of paresis with marked meningeal changes as shown in the fluid formula a decided temporary improvement and apparent arrest of the process can be effected by intensive intravenous and intraspinal treatment. The meningitis and superficial lesions in the cortex are reached by the remedies. The deeper lesions remain quiescent for a time and again become active. Patients die with advanced brain atrophy with little or no evidence of meningitis. The chances of arresting the pathological process in tabes are much greater than in paresis. The treponemata are more accessible to our drugs as the secondary degenerations prob-

ably originate from a meningitis about the posterior nerve roots. A progressive tabes shows in the fluid the cytology of a meningitis. The process has in many cases been permanently arrested by combined intravenous and intraspinal medication. It is useless in advanced degenerations with little or no evidence of a meningitis. I have in no manner changed my opinion as to the great value of properly employed intraspinal medication in properly selected cases in spite of the theoretical objections that have been advanced by its opponents. Cases treated years ago have returned for control observation and puncture. Their fluid findings continue negative and no clinical signs point to progress in their cord degeneration. The method has stood the test of time and is a valuable adjunct to our therapeutic measures. It is well to again emphasize the relationship of the fluid findings in early syphilis to these late degenerations. If recognized early and properly treated both tabes and paresis will theoretically cease to exist. The neurologist sees the end-stages of tabes and paresis and pronounces them incurable; the syphilologist should recognize their early beginnings and cure them. Ability to visualize the pathological changes in syphilis from the primary sore to the dissolution of the most specialized and highest brain centers gives one much respect for the possibilities of the invading organism. In prognosticating the fate of the infected individual the defensive forces of the body should always be kept in mind. Every syphilitic is not a potential paretic or tabetic; neither must he necessarily die from the cardiovascular phase of his infection.

From the experimental view point we know little of the defensive forces of the body with the exception of the fibrosis which localizes and extinguishes the focal lesions and the attenuation of the virulence of the infectious agent by time. The usual immunizing substances cannot be demonstrated in syphilitic blood or tissues. Spontaneous cure may, however, occur in syphilis without treatment and many cures followed treatment in the pre-arsphenamine era by methods now regarded as inefficient.

In congenital syphilis the result of recent infection in the parent, early abortion in the first pregnancy is followed by a syphilitic child born at term and later by children who are born apparently healthy but who develop interstitial keratitis or other phases of late congenital syphilis.

In these cases which give us a clinical demonstration of an attenuation of the virus we must assume that the mother develops antibodies which are transmitted to her child with the organisms of the disease. In prognosticating the future of the syphilitic child one should therefore regard the age of the infection in the parents. Children born of parents who acquired the infection several years before marriage may escape it entirely but many such children who present a perfect physical condition on investi-

gation will be found to have positive Wassermann reactions. I have in mind a man who married eight years after his infection—who received what at that time was considered adequate treatment. He consulted me for a sudden deafness in his four-year old child. Both the blood and spinal fluid showed positive Wassermann reactions. A six-year old daughter also had a 4 plus reaction in the blood, the cause of which was not revealed by examination. Both the children were well developed mentally and physically. The existence of the infection would not have been suspected had it not been for the sudden onset of the eighth nerve involvement.

From observations of cases like these, which are not unusual, it would seem advisable to restudy the entire subject of congenital syphilis and to carefully control the possibility of the disease by serological tests at various times after birth. We are at present treating at the Vanderbilt Clinic a large number of congenital syphilitic infants and, where possible, investigating their spinal fluids. This is done usually after treatment and our statistics, while not yet tabulated, give the impression of a similar incidence of involvement as in the adult. We have obtained results in these infants by intramuscular injections of mercury and neoarsphenamine, which are encouraging both as to the clinical manifestations and the blood reactions, and feel that we can offer a more hopeful prognosis than by the use of mercury internally or by inunction. It is not yet possible to say how much, if at all, we can influence the nervous system involvement by the method in question. Intraspinal treatment in the small patients has not yet been attempted by us because of the technical difficulties and the possibility of damage to the delicate structures of the infant. We shall probably see the usual number of cases of juvenile tabes, paresis or other types of neurosyphilis as in the adult whose early neurosyphilis has been overlooked or treated improperly.

The later phases of congenital syphilis, such as interstitial keratitis, yield much more rapidly to arsphenamine than to mercury and cease to relapse when the blood reaction becomes negative.

It is possible in many cases of congenital syphilis by persistent and well directed treatment to change what was considered a Wassermann fast reaction.

In acquired syphilis many cases are met with which give a persistent and strongly positive Wassermann reaction with and without clinical symptoms. In 90 per cent of these cases which came to autopsy at the City Hospital the aortic walls showed focal and advanced lesions of syphilis. In some of the cases the physical signs of aortic disease were not present during life. Warthin's observations that all syphilitic patients who come to autopsy have small focal lesions in the heart, muscle or viscera explains many of the mysteries of the so-called Wassermann fast cases. The lesions are often too small to cause symptoms and the diagnostic skill of the clinician cannot uncover them. A persistent Wasser-

mann reaction in the blood or one which disappears under treatment and slowly returns during rest periods may be due to reinfection of the blood from the central nervous system. I have met with instances of this kind where the reaction disappeared permanently after intraspinal treatment of a neurosyphilis. The majority of persistent reactions can be altered by their interpretation and properly directed treatment. In paresis we have an example of what may properly be termed a Wassermann fast reaction in the spinal fluid. It persists during years of intensive treatment. The blood Wassermann may be modified but in the fluid seldom or never. Frequent changes in this reaction in the fluid in well developed cases of neurosyphilis we seldom see. In types of early syphilitic meningitis we have exceptionally met with these fluctuations. In curable cases of neurosyphilis the various phases of the fluid yield in regular order and gradually, and not by up and down gradations.

In discussing some of the factors which bear on the prognosis of syphilis I have endeavored to show that the infection in the late stages may be localized in organs with a large margin of safety or in tissues whose impairment does not endanger life. Late lesions in the skin may exist and recur for years with no impairment of the general health. The small visceral lesions described by Warthin are met with in organs which may withstand for years their slow progression and ultimately fibrosis may end their activity. These favorable factors should not be overlooked nor should they lead us to minimize the effects of the disease when it involves the heart, the large vessels, the nervous system and the eye.

The individual syphilitic should be regarded as a potential candidate for the more serious phases of the infection and should be safe guarded as far as our modern diagnostic and therapeutic measures are available. The majority of the intelligent class of patients will coöperate with the physician who handles them tactfully and informs them of the reason for each procedure. The results of treatment and the justification of a favorable prognosis rests mutually on the physician and the patient.

**Summary.** In summarizing we might say the future of the syphilitic patient depends upon the knowledge and training of the physician first consulted. If familiar with modern diagnostic methods he will not neglect the employment of the dark-field microscope. A search for the treponemata may be rewarded by an examination of the fluid aspirated from the communicating lymph nodes after failure to demonstrate the organism in the primary sore. When the diagnosis is made the physician should impress the patient with the necessity for vigorous and thorough modern treatment and endeavor to make him appreciate the seriousness of the disease.

Mercury by mouth is a desultory way is of no value in aborting the infection. Many patients receive insufficient treatment

because physicians have not acquired accurate knowledge of the modern specific drugs and are ignorant of their proper administration. This criticism is as valid now with our greater knowledge as formerly before our modern studies of the disease. The fault today lies not only in the lack of training on the part of the physician but also in his failure to impress the patient with the necessity of continuing treatment long after the symptoms have disappeared. All physicians treating syphilis should have a thorough knowledge of the pathology of the disease and its possibilities in the early as well as the late stages.

In the primary stage, before the Wassermann reaction becomes positive a cure generally may be assured by intensive treatment. At least two courses of 8 doses each of arsphenamine and two courses of 15 injections each of mercury should be given. Continued observation and control by the Wassermann test and lumbar puncture are necessary as in the treatment of later stages.

In the secondary stage, after the Wassermann reaction has become positive, a cure is not so readily obtained but is possible if treatment is thorough and not stopped as soon as the blood becomes negative. The positive reaction continues as a rule for some time after all the other symptoms have disappeared. Depending on the length of time the blood has been positive, patients should receive from two to three courses of arsphenamine and three or more courses of mercury. If the spinal fluid is positive, more treatment may be required, including intraspinal injections. Cure should be pronounced only after a long period of observation. A guarded prognosis should be given when the disease has progressed beyond the early secondary stage.

A symptomatic cure is possible in latent and tertiary stages if adequate treatment has been given, even in so-called "Wassermann-fast" cases. Adequate treatment means the administration of not less than two to four courses of arsphenamine combined with and followed by mercury, iodides and mixed treatment. In the so-called "Wassermann-fast" cases an examination of the spinal fluid is imperative.

The prognosis is not favorable in certain types of neurosyphilis. This statement applies especially to the degenerative stages of tabes and paresis. It is, however, possible to anticipate and arrest the progress of early active tabes and paresis. The majority of cases do not respond to treatment as ordinarily administered. Early cases of neurosyphilis which do not respond to intravenous treatment combined with mercury are generally cured by intraspinous treatment. Paresis and tabes result from neurosyphilis improperly treated in the early stages.

In congenital syphilis, with the old methods of treatment it was impossible to obtain a negative Wassermann reaction and even now the prognosis is not as favorable as in the acquired disease because of the severity of the infection, interference in the develop-

ment of the growing tissues and the difficulty of carrying out the treatment.

In congenital syphilis with involvement of the central nervous system, because of the technical difficulties in treatment the prognosis is less favorable than in the similar type of syphilis in adults.

It is frequently necessary to give a prognosis because of contemplated matrimony. While a rigid standard cannot be maintained physicians should endeavor to obtain a negative Wassermann reaction in the blood for one or two years after adequate treatment, with a negative spinal fluid, before giving consent. The patient should remain under observation for several years. If the patient be a woman with a positive Wassermann reaction treatment should be begun during early pregnancy and continued throughout.

Marriage may be permissible in spite of a positive blood Wassermann if thorough treatment has been administered. In exceptional cases after prolonged treatment and after the expiration of at least five years from the time of the infection and two years' freedom from all symptoms, a conditional consent to matrimony may be given.

The marriage of heredo-syphilitics with the disease still active is not desirable, even though the danger of transmission of infection to the third generation is negligible.

We must remember that untreated or imperfectly treated syphilis is essentially a relapsing disease. In thoroughly treated cases, however, the prognosis is favorable.

The modern criteria of cure are dependent on: (1) Adequate treatment; (2) a negative Wassermann reaction, for at least a year after cessation of all treatment, which continues negative after a provocative injection of arsphenamine; (3) a negative spinal fluid; (4) negative findings in the cardiovascular system. The early involvement of the cardiovascular apparatus may be followed by changes in the heart and aorta with a negative Wassermann reaction.

In this paper, which is more or less fragmentary and incomplete, many factors which influence the prognosis of syphilis have not been touched upon. I would like to suggest to those who treat syphilis to acquaint themselves more accurately with the possible toxic effects of the modern arsenicals. Has the hypertension which is at times met with after prolonged treatment any relation to the known toxic action of arsenic on the bloodvessels? How long is arsenic stored in the liver after the cessation of our usual courses of the drug? Can jaundice be prevented by a lno protein diet during treatment? Is the elimination of arseicw hastened by the simultaneous use of the iodides? To what extent do our various methods of giving arsphenamine develop arsenic-fast strains of the treponemata and thus render our further therapeutic attacks less efficient?

# THE HEMOLYTOPOIETIC SYSTEM IN THE PRIMARY ANEMIAS, WITH A FURTHER NOTE ON THE VALUE OF SPLENECTOMY.[1] [2]

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It is convenient and desirable, from a practical point of view, to consider all diseases of the blood, but especially the so-called "primary anemias," from the dynamic standpoint of the constant interplay of the blood-forming and blood-destroying apparatus (which may be termed the hemolytopoietic system and the adjustment thereof spoken of as the hemolytopoietic balance). By using the various diagnostic measures now available there can thus be determined not only the actual disease in question, but whether it is chiefly due to an excess of blood destruction or to a paucity of blood formation; and in the individual case, how grave a disturbance of this hemolytopoietic balance exists and whether it is becoming more or less upset.

**Hemolytopoietic Balance.** A correct estimation of the condition of this mechanism at a given moment (practically the hematopoietic-hemolytic index of Schneider[3]) is rendered difficult, both by the semiquantitative nature of some of the tests required and by the complexity of the system itself. To discuss the latter point first, a steadily accumulating mass of evidence is forcing us to recognize that bone-marrow, lymph nodes, spleen, liver and the whole "reticulo-endothelial apparatus"[4] must be considered as definite a mechanism for the control of the cellular elements of the blood as the digestive or endocrine systems are in their respective spheres. As with the latter system, the complexity of the problem is made more acute by the marked (and as yet imper-

[1] Read before the New York Academy of Medicine, January 18, 1923, and in abstract before the Association of American Physicians, May 1, 1923.

[2] The "hemopoietic system," as it has been loosely designated, is not a desirable term, as it ignores the blood-destroying mechanism, which is at least of equal importance with that of blood formation. Custom has long sanctioned the use of "hemo" rather than "hemato," in the compound word.

[3] Journal-Lancet, 1917, **37**, 105; Arch. Int. Med., 1916, **17**, 32; ibid., 1917, **19**, 156.

[4] By "reticulo-endothelial apparatus" is meant the widespread cells of reticular or endothelial origin that possess marked capacity for phagocytosis of foreign bodies, body cells, bacteria, etc. They are similar to or identical with the macrophages of Metchnikoff, klasmatocytes of Ranvier, adventitia cells of Marchand, resting wandering cells of Maximow, pyrrhol cells of Goldmann, endothelial leukocytes of Mallory, histiocytes of Aschoff. Jaffé (Wien. klin. Wchnschr., 1922, **35**, 595) has shown that they are functionally significant as the Kupffer cells of the liver, the pulp cells (splenocytes of the spleen in the endothelium of spleen, lymph nodes, bone-marrow and adrenal and reticulum of spleen, lymph nodes and bone-marrow.)

fectly delimited) ability of one member of the system to take over, in times of stress, functions belonging primarily to some other member of the group. Thus, where blood destruction is greatly increased (as in hemosiderosis, some cases of pernicious anemia and chronic malaria) or in experimental plethora,[5] or after the administration of hemolytic immune serum,[6] the products of blood destruction are found in Kupffer cells, lymph nodes and bone-marrow (parts that ordinarily are not noticeably concerned in this process) in much larger quantities than in other parts of the body. The quick response of the normal bone-marrow to demands for more blood cells is well known, but, in addition, if sufficiently intense and protracted strain is put upon the blood-forming apparatus, it is not infrequent to find other members of the hemolytopoietic system reverting to their embryonal functions or metaplastically taking on myeloid activity;[7] and even in a case I recently reported, the bone-marrow assumed definite lymphopoiesis.[8] By quite similar reasoning is to be explained the adjustment that follows splenectomy in normal individuals, whereby after a relatively short period of time the splenectomized patient or animal returns practically to normal in most or all hematological details.

**Methods Available.** Reverting to what I have spoken of as the semiquantitative nature of some of the tests involved, I would like to review what evidence may be obtained toward estimating the amount of blood-cell formation and destruction. Although this involves statements that are far from new, I hope that the repetition will be pardoned, in view of the fact that apparently in few clinical laboratories or in few reported cases in the literature has full advantage been taken of available methods.

On the side of blood destruction is the evidence to be obtained from the appearance of the erythrocytes when vitally stained or in stained spreads, and, more directly, from the urobilin output in feces or duodenal contents. According to current views,[9] erythrocyte destruction begins while the cells are still in the circulation. By a process of fragmentation they become smaller and smaller until the disintegrated products are taken up as hemoglobin-bearing dust chiefly by the spleen, to be turned over to the liver by steps that we know little about, there to be excreted

[5] Krumbhaar, E. B., and Chanutin, A.: Jour. Exp. Med., 1922, **35**, 8427.

[6] Pearce, R. M., Krumbhaar, E. B., Frazier, C. H.: The Spleen and Anemia, Lippincott, 1917, p. 180.

[7] Donhauser, J. L.: Bull. Ayer Clin. Lab., 1908, **5**, 46. Bunting, C. H.: Jour. Exper Med., 1906, **7**, 365. Dominici: Arch. de méd. expér. et d'anat. path., 1901, **13**, 1. Domarus: Arch. exp. Path. u. Phar., 1908, **58**, 319. Tanaka: Ziegler's Beitr., 1912, **53**, 388 (Lit).

[8] Krumbhaar, E. B.: Jour. Med. Research, 1922, **43**, 369.

[9] Rous, P., and Robertson, O. H.: Jour. Exper. Med., 1917, **25**, 651 and 665.

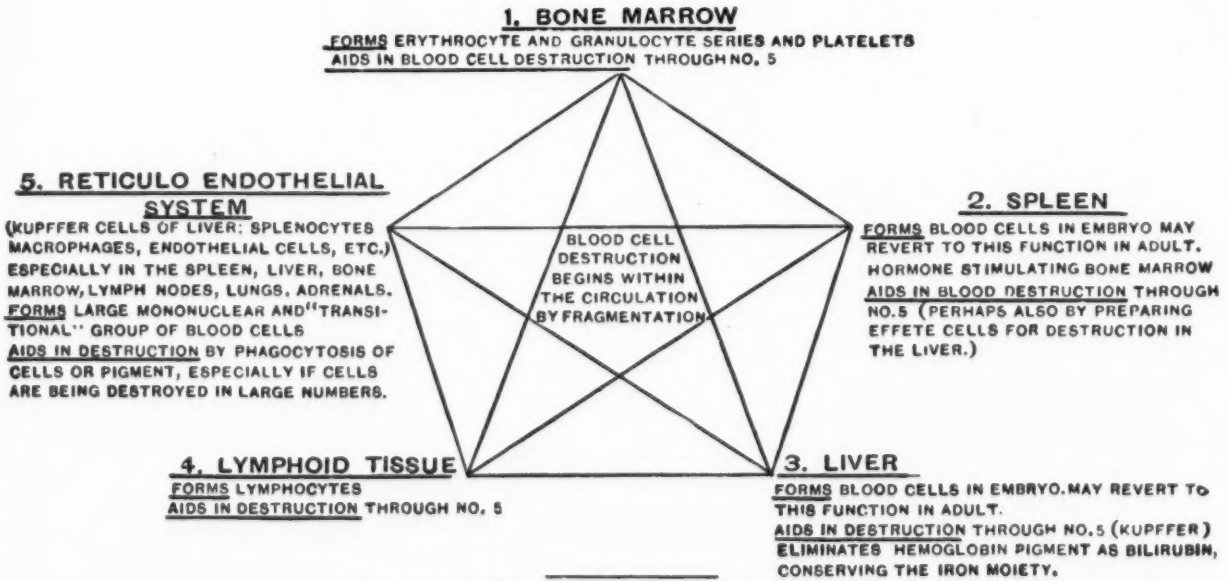
1. BONE MARROW
FORMS ERYTHROCYTE AND GRANULOCYTE SERIES AND PLATELETS
AIDS IN BLOOD CELL DESTRUCTION THROUGH NO. 5
2. SPLEEN
FORMS BLOOD CELLS IN EMBRYO MAY REVERT TO THIS FUNCTION IN ADULT.
HORMONE STIMULATING BONE MARROW
AIDS IN BLOOD DESTRUCTION THROUGH NO. 5 (PERHAPS ALSO BY PREPARING EFFETE CELLS FOR DESTRUCTION IN THE LIVER.)
3. LIVER
FORMS BLOOD CELLS IN EMBRYO. MAY REVERT TO THIS FUNCTION IN ADULT.
AIDS IN DESTRUCTION THROUGH NO. 5 (KUPFFER)
ELIMINATES HEMOGLOBIN PIGMENT AS BILIRUBIN, CONSERVING THE IRON MOIETY.
4. LYMPHOID TISSUE
FORMS LYMPHOCYTES
AIDS IN DESTRUCTION THROUGH NO. 5
5. RETICULO ENDOTHELIAL SYSTEM
(KUPFFER CELLS OF LIVER; SPLENOCYTES MACROPHAGES, ENDOTHELIAL CELLS, ETC.) ESPECIALLY IN THE SPLEEN, LIVER, BONE MARROW, LYMPH NODES, LUNGS, ADRENALS.
FORMS LARGE MONONUCLEAR AND "TRANSITIONAL" GROUP OF BLOOD CELLS
AIDS IN DESTRUCTION BY PHAGOCYTOSIS OF CELLS OR PIGMENT, ESPECIALLY IF CELLS ARE BEING DESTROYED IN LARGE NUMBERS.
BLOOD CELL DESTRUCTION BEGINS WITHIN THE CIRCULATION BY FRAGMENTATION

FIG. 1. HEMOLYTO-POIETIC SYSTEM

as bile pigment with conservation of the iron fraction. Microcytes, therefore, in the peripheral blood, and poikilocytes as well, are to be taken as evidence of increased blood destruction. This has clinical confirmation in hemolytic jaundice—the disease *par excellence* of excessive blood destruction, where microcytes are notoriously numerous. However, Price-Jones[10] has recently showed that whereas in pernicious anemia the average size of the red cell is greater than normal, after hemorrhage it is smaller than normal—a sign, therefore, of deficient blood formation. The degenerated "stippled" erythrocytes of lead poisoning are not within the scope of this paper.

The urobilin output should give us direct evidence of the amount of blood destruction, but unfortunately hitherto unsurmountable difficulties leave this still as a semiquantitative test only. If the original technic of Wilbur and Addis[11] is followed, not only is the test a fairly lengthy one, but the unknown factor of the amount of urobilin absorbed by the intestine cannot be accounted for. In Schneider's[12] method of using a sample of duodenal juice, normal fluctuations of output, as well as obvious difficulties in collecting a valid sample, present to my mind even greater handicaps. In addition, there has been no standardization of several factors such as intensity of light, width of column of fluid and other details which may seriously affect the reported result; and in Todd's book, *Clinical Diagnosis* (1919), and Haden's *Clinical Laboratory Methods* (1923), the only text-books I can find in which the test is mentioned at all, calculations apparently give a result ten times greater than Wilbur and Addis intended.* This state of affairs is so regrettable that work is now going on in our laboratories to make this test serviceable by eliminating preventable errors. Fortunately, the different results obtained in hyperhemolysis or hypopoiesis (as compared with the normal) are so great that, in spite of the difficulties first referred to, I am confident the test will prove to be of sufficient value to be used in every carefully studied case of anemia.

On the side of blood formation, information can be obtained from the blood counts (hemoglobin, erythrocytes, leukocytes, platelets), especially if frequently repeated accounts are made; but care should be taken not to place reliance on variations so slight that they are within the limits of observational error. The less constant the conditions attending the various tests and the less your acquaintance with the source of the report the greater should be your conservatism in this respect. Especially valuable information may be obtained from a properly prepared vital or stained spread. Nucleated forms are a sure indication of bone-

[10] Jour. Path. and Bacteriol., 1922, **25**, 487.

[11] Arch. Int. Med., 1914, **13**, 235.

[12] Loc. cit.

* Details of Giffin's modification of the method of performing the test on the duodenal contents can be found in Simon's Clinical Diagnosis, 10th ed., p. 347.

marrow activity, and quantitative comparisons may, therefore, be used to estimate roughly both the stress of the demand and the ability to supply. The normoblast may appear in any severe anemia; the megaloblast, or earlier form, is more rarely found, except in pernicious anemia, where it possesses diagnostic value. Nuclear remnants, Heinz bodies, Howell-Jolly bodies and Cabot rings are similar signs of bone-marrow activity. In terminal conditions, however, many and bizarre nucleated forms may appear in the blood streams without finding any bone-marrow hyperplasia at autopsy. The size and shape of the erythrocytes have been already referred to; numerous macrocytes, presumably coming from megaloblasts, suggest pernicious anemia. Extensive polychromatophilia (young erythrocytes) also indicates bone-marrow activity, though this is better brought out by the special search for reticulocytes in "vital spreads."[13] Like the urobilin test, this simple test deserves much more frequent use than has yet been accorded it. It is not only a sensitive method of ascertaining and following the bone-marrow activity in a given case, but in hemolytic jaundice, where for some unexplained reason there is a marked reticulosis, it assumes diagnostic value. The shower of nucleated forms and increased number of reticulocytes and platelets in pernicious anemia following splenectomy and at the beginning of a remission are both well-known phenomena.

Finally, the test for the resistance of erythrocytes to hypotonic salt solution[14] determines this phase of blood-cell formation and indirectly throws light on the diagnosis and severity of the process. In hemolytic jaundice, essentially a disturbance in the resistance of the erythrocytes, this test is pathognomonic.

**Results of Treatment.** Treatment of any anemia is naturally directed toward relieving the anemia and removing the cause. The best methods of securing the former of these factors cannot be discussed here, while the attainment of the latter is obviously apparent in secondary anemias. It is in the so-called "primary anemias," however, where the causes are unknown, that the most earnest attempts have been made to restore the lost hemolytopoietic balance by direct intervention with one or other member of the system. Removal of the spleen, one of the chief agents of blood destruction, has proved valuable to a greater or less degree in the several diseases of this group, the value of the procedure always being due to removing one of the chief agents of blood destruction. Although, as is shown in the diagram I have presented, this function can be and is taken over by other members of the hemolytopoietic system it is not exerted to such a great

[13] Vogel, C.: Arch. Int. Med., 1913, **12**, 707. Krumbhaar, E. B.: Jour. Lab. and Clin. Med., 1922, **79**, 1879, etc.

[14] Pearce, R. M., Krumbhaar, E. B., and Frazier, C. H.: The Spleen and Anemia, Lippincott, 1917.

extent for a considerable period, and in some case not ever. According to the doctrine of "hypersplenism," the diseased spleen has developed this function to a pernicious extent, a condition which may not be attained by the other members of the system after splenectomy.

In considering in a more detailed manner the effects of splenectomy in individual diseases, the condition has not materially changed since I last collected reports on this question.[15] [16] Nevertheless, by a swing of the pendulum, which is so common in medical advances, splenectomy for pernicious anemia, the most important disease of the group, is now, in my opinion, being neglected just as much as it was being overdone six years ago. The conclusions that were reached at that time have, it seems to me, largely been confirmed by subsequent studies. The operative mortality has been lowered, and most of the survivors have shown marked temporary improvement, the remission usually lasting longer than the average length of natural or post-transfusional remissions. In fact, there is a respectable number of cases that have continued in good health for two to six years after splenectomy. Therefore, my former question, "May it not develop that a larger percentage of such cures (3 in 1200 by the older methods, according to Cabot) will follow splenectomy even though the blood picture does not return absolutely to normal?" can, I think, properly be answered in the affirmative. Of the 208 cases in my list, Giffin reports that 11 of his 53 lived more than three years after operation and 5 still live between four and five years after operation. Of Walterhofer's 16 cases, 1 survived for five years, and 2 more for from two to four years, and so on. This does not mean that every case of pernicious anemia should have the spleen removed or that a cure should ever be hoped for; but it does, to my mind, indicate that in certain cases, especially those that have a relatively recent onset, with a fair blood picture, signs of an enlarged spleen and increased hemolysis, splenectomy is usually advisable. How it should be combined with transfusions is a question that I cannot take up at this time.

In hemolytic jaundice the results of splenectomy continue to be excellent. Although in many cases the resistance of the erythrocytes does not return to normal (indicating that the primary cause of the disease is not in the spleen but probably in the erythrocytes themselves), nevertheless the anemia and jaundice disappear, the urobilin excretion and reticulocyte percentage diminish and the patient is for practical purposes well. If Mayo's caution not to operate during a "crisis of deglobulization" is heeded, the operative mortality may be reduced even below the present rate of 3 per cent.

[15] Krumbhaar, E. B.: Trans. Coll. Phys., Philadelphia, 1916, **38**, 158.
[16] Krumbhaar, E. B.: Pernicious Anemia, Jour. Am. Med. Assn., 1916, **67**, 723.

The improvement that has long been known to follow splenectomy in Banti's disease is supported by the later figures. Abdominal adhesions and hemorrhage from varicose veins add to the difficulties of the operation, but marked improvement can be promised to most of the survivors. In fact, many have continued so long without symptoms that they have been reported as cured, but it is unwise to use the term "cure," when the operative procedure has merely removed the chief disturber of the hemolytopoietic balance rather than the cause of the disease itself. I, of course, recognize that as negative methods are the only ones at present available for diagnosing Banti's disease, many cases of splenomegaly due to lues, cirrhosis, portal phlebitis and similar causes are undoubtedly included, and it is especially hard to distinguish them when successful removal of the spleen in the latter group also results in improvement. Nevertheless, the conviction persists that Banti described a primary disease the cause of which is unknown, but which is greatly improved by removal of the spleen.

The most important additions that have been made to our knowledge of this subject in recent years have come from the Mayo Clinic.[17] [18] Thanks to them not only have most of our previous opinions been confirmed, but reliable estimates made about the value of splenectomy in newer fields. Thus while leukemia was formerly considered as definitely contraindicated on account of the high operative mortality (see Table), the Mayos have had only 1 postoperative death in 26 cases (much less than in splenic anemia); on the other hand, 15 have subsequently died, so that Giffin considers splenectomy helpful only in a few of the very chronic types of myelogenous leukemia where the organ is fibrous and the leukocyte count never very high. Splenectomy in cases of splenomegaly associated with chronic sepsis has also proved unsatisfactory, both on account of the high operative mortality (20 per cent) and the poor end-result (3 out of 10 survive, and 1 of these is in poor condition). In luetic and malarial splenomegaly not only are the patients' conditions directly improved by removal of the spleen, but they are also usually rendered much more amenable to specific treatment, to which they may previously have proved intractable. In fact, if I may venture an opinion on what is more directly a surgical problem in such secondary splenomegalies, the question being mainly one of operative risk, it would seem desirable to limit the operation to chronic cases that have proved resistant to all other forms of treatment and to proceed with extra conservatism where many adhesions multiply the chances of shock and hemorrhage. For the latest

[17] Mayo, W. J.: Jour. Am. Med. Assn., 1921, **77**, 34.
[18] Giffin, H. Z.: Minnesota Med., 1921, **4**, 132.

brilliant success of splenectomy, namely in the hemorrhagic diatheses, the reader is referred to Brill's communication.

**Summary.** 1. Bone-marrow, spleen, liver, lymph nodes and reticulo-endothelial apparatus combine to regulate the formation and destruction of blood cells in what may be termed the "hemolytopoietic system."

2. The methods for studying the functioning of this system ("hemolytopoietic balance") are discussed.

3. The results of splenectomy in various diseases affecting this balance are presented.

RESULTS OF SPLENECTOMY IN MEDICAL DISEASES.*

| Disease. | Number of cases | Postoperative death (within 1 month). | Postoperative mortality. | Record of subsequent death. | Unimproved. | Improved. | Apparently cured. |
|---|---|---|---|---|---|---|---|
| Splenic anemia | 293 | 40 | 13.6 | 24 | 4 | 178 | 35 |
| Gaucher's disease | 16 | 4 | 25.0 | 1 | .. | 9 | 1 |
| v. Jaksch's disease | 19 | .. | .. | 1 | .. | 11 | 1 |
| Hemolytic jaundice | 137 | 4 | 2.9 | .. | .. | 5 | 125 |
| Pernicious anemia | 208 | 35 | 16.8 | 79 | 26 | 144 | .. |
| Myelogenous leukemia† | 80 | 47 | 56.7 | 21 | 2 | 7 | .. |
| Liver cirrhosis | 19 | 6 | 31.6 | 5 | 4 | 7 | .. |
| Luetic splenomegaly | 7 | 1 | 14.3 | .. | .. | 6 | .. |
| Tuberculous splenomegaly | 17 | 2 | 11.7 | 2 | 1 | 11 | .. |
| Malarial splenomegaly | 250 | 48 | 19.2 | .. | .. | 202 | .. |
| Hemorrhagic diathesis | 27 | 2 | 7.4 | .. | 1 | 9 | 15 |
| Total | 1073 | 190 | 17.6 | 133 | 38 | 591 | 177 |

† The postoperative mortality in recent reports of this disease is no higher than the average of other diseases of the list.

* Although larger than any figures heretofore published on this topic, this table by no means attempts to be a complete list. It has been compiled from reports from the following authors:

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# THE CAUSE OF BOTHRIOCEPHALUS ANEMIA. (REPORT OF A CASE.)

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The broad tape worm, bothriocephalus latus, is very commonly encountered in the Baltic provinces of Russia, the Scandinavian countries, and in certain parts of Switzerland. It is the most common tapeworm of China and Japan. The statement[1] is made that 20 per cent of the people living around Lake Geneva are infested and that 9 per cent of the inhabitants of Moscow harbor the parasite. The intermediate host is the fish. Man is infested by eating raw or improperly cooked fish containing the cysticercus stage of the parasite.

Bothriocephalus infestation is distinctly unusual in the United States. Cases are of more than passing interest on account of their rarity alone. Singer,[2] in reporting a case, was able to find only 30 cases reported in this country up to 1916. Since that time single cases have been reported by Rubenstone,[3] Kopelowitz,[4] McGoffin,[5] Levy and Wall,[6] Savitz,[7] and Wallace and Grant,[8] and 6 cases by Becker.[9] Of the 30 cases found by Singer reported before 1916, only 1 had originated in the United States. One of the cases reported by Becker and the case reported by Wallace and Grant were native born. It is evident that the fish in certain districts of this country must be infested. If so, it is only a matter of time before more cases will be reported as originating here.

Clinicians are interested in bothriocephalus infestation not because of its rarity, but by reason of the anemia which may occur in the infested individual. It is said that about 10 per cent of those harboring the parasite at some time develop an anemia. In a considerable percentage of cases the anemia cannot be distinguished clinically from true idiopathic pernicious anemia, thus differing from the secondary type of anemia occurring in other kinds of parasitic infection.

This then is one of the few conditions in which there is an anemia of known etiology which so closely simulates pernicious anemia. It has for this reason excited the interest of many research workers in the hope that the solution of the cause of the anemia in bothriocephalus infection might throw some light on the origin of pernicious anemia.

The cause of the primary type of anemia in this disease has been variously explained. It has been thought by some that certain lipoids of the worm might be the hemolytic agent. Attempts to reproduce the anemia with extracts of the normal worm have not

been successful. Schaumann and Tallquist[10] found however, that by feeding or injecting subcutaneously dogs with worms which had been subjected to tryptic digestion an anemia could be produced. In one dog the anemia was fatal, in the other the red-cell count was reduced one-half. They found also that similar extracts were hemolytic *in vitro* for blood of the dog.

Faust and Tallquist[11] noted that the worms recovered from cases having an anemia show as a rule a great loss of substance. There was sometimes almost a complete solution of the proglottides. They found that on an average there was a 60 per cent loss of dried substance per unit of length as compared with the normal worm. There was little change in the lipoid content. The cause of the disintegration was not clear. They advanced the theory that through death of the parasite in the intestinal canal of the host hemolytic substances were absorbed and brought about the characteristic action on the blood and bone-marrow. The time and extent of the absorption would determine whether or not there is an anemia, the anemia developing only when there is a relative insufficiency of the bone marrow.

These observations of Faust and Tallquist seem to have received little recognition. Their theory is seldom mentioned in a discussion of the cause of bothriocephalus anemia. We have had the opportunity of studying a case of bothriocephalus infestation in which there is much to suggest that the onset of the anemia was due to the death of the worm. The case history is as follows:

**Case History.** F. A., a laborer, aged thirty-two years, admitted to the Henry Ford Hospital, March 23, 1921.

*Complaint.* Dizziness, pain in leg, weakness and difficulty of vision.

*Family History.* Unimportant.

*Personal History.* The patient had come to this country from Finland in 1914. He stated that he had had none of the usual childhood diseases. He had had three attacks of pneumonia. He gave a history of nycturia. He had passed segments of a tapeworm at intervals for the past three years. Otherwise he had been entirely well.

*Present Illness.* Five weeks before admission he began to have pain and swelling in the right knee and ankle. The pain was present only on motion. At the same time he noticed a small red papular eruption over both legs. Soon after a large purple area appeared on the inner surface of the left knee. He continued to work, but became progressively weaker. For a week he had had some dimness of vision and had been dizzy on exertion for two weeks. He complained of an indefinite pain in the epigastric region and had vomited once.

*Physical Examination.* The patient was evidently ill. There was a slight fever; the pulse was 96. There was no glandular

enlargement. Examination of the head, lungs, heart and abdomen showed no abnormality. The blood-pressure was 120/70.

Over the thighs, legs and to a less extent over the back, there were numerous petechiæ. There was a very large ecchymosis about the left knee and down the left leg. Purpuric spots were present on the uvula and hemorrhages were found in the retina.

The left knee was painful on passive motion. There was some tenderness on pressure at the medial margin of the left patella. No fluid could be demonstrated in the knee-joint. Both knees subsequently showed a yellowish green discoloration in the subcutaneous tissues. The appearance suggested a sanguineous effusion in the joint. The gums were spongy and hemorrhagic.

*Laboratory Findings.* Urine: Specific gravity 1012 to 1028, albumin and sugar negative, microscopical examination negative. Blood count: Red blood cells, 2,664,000; white blood cells, 4950; hemoglobin 50 per cent; differential count: neutrophiles, 69 per cent; eosinophiles 0; basophiles, 1 per cent; small mononuclears, 25 per cent; large mononuclears, 5 per cent. Wassermann negative. Blood culture negative. Examination of the stool revealed large numbers of the ova of bothriocephalus latus.

*Subsequent Course.* Two days after admission the red cell count had dropped to 2,000,000 and the hemoglobin to 40 per cent. As soon as ova were discovered in the stool, treatment was instituted. A specimen of bothriocephalus latus approximately 35 feet in length was passed. The ecchymosis and purpuric spots disappeared. The joint signs and symptoms cleared up. The anemia began to improve at once. Sixteen days after admission the red cell count was 3,673,000, and ova could no longer be found in the stool. The patient was discharged after a stay in the hospital of twenty-five days.

**Discussion.** The patient had resided in this country six years. He had come from Finland where infestation with bothriocephalus latus is exceedingly common. In all probability he had acquired the infection before coming to the United States. He had passed segments of a tapeworm at intervals for at least three years. There is every reason to believe that the worm had been in the intestinal tract for at least six years.

The patient had remained perfectly well however, until the sudden onset of the present illness. On recovering the worm its segments were found to be densely intertwined. A large part was glistening and gelatinous, and evidently undergoing disintegration. The rest of the worm looked perfectly normal. The parts of the worm were so intertwined that the decomposing parts could not be cast off until the dissolution was complete.

These findings are in accord with Faust and Tallquist's observation that the worms recovered from patients with an anemia, show a great loss of substance. The history and the findings suggest

that the onset of symptoms in this case was due to the death of the worm and the anemia, due to absorption of decomposition products.

**Summary.** A case of bothriocephalus latus infestation is reported. The patient had probably harbored the parasite for six years without showing any symptoms. There was an anemia of sudden onset which quickly disappeared after the worm was expelled. When recovered a large part of the worm was in a state of decomposition. These findings lend support to the idea expressed by Faust and Tallquist that the anemia of bothriocephalus infestation is due to the absorption of hemolytic substances from the dead worm.

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## CEREBROSPINAL FLUID PRESSURES: CONCERNING AN INITIAL FALL IN PRESSURE READINGS AND THE METHOD OF OBTAINING A STANDARD READING.[1]

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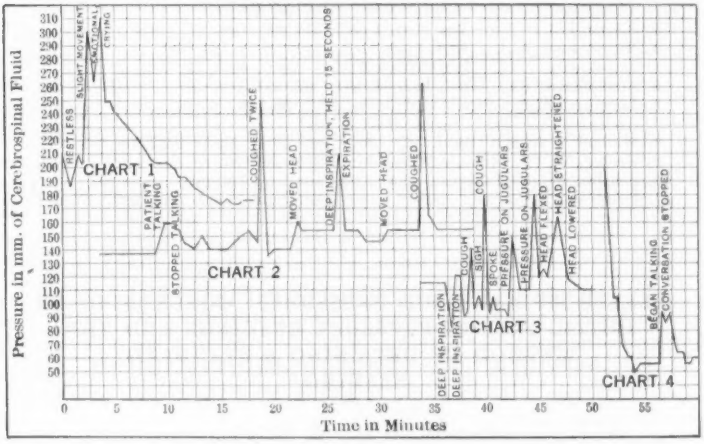
The reading of the cerebrospinal fluid pressure manometer taken shortly after a lumbar puncture has been performed does not represent the cerebrospinal fluid pressure that exists under conditions of rest and equilibrium. The reading immediately following the lumbar puncture may be 300 or 400 per cent or more in excess of the pressure that will be obtained at the end of five to ten minutes or thereabouts, and this latter pressure will often remain constant as long as the patient is quiet and does not move.

[1] This work was greatly assisted by a grant of funds from the United States Interdepartmental Social Hygiene Board.

The ordinary method of taking the cerebrospinal fluid pressure is to perform a lumbar puncture with the patient lying on his side on a level table. A manometer is then attached to the lumbar puncture needle and the reading made of this manometer. Either a mercury manometer or a standpipe recording the height to which the cerebrospinal fluid rises may be used. In either case the principle is quite the same and should represent the tension of the cerebrospinal fluid at that moment. In our experiments a standpipe manometer was used to record the fluid pressure. The point we wish to emphasize is that the tension of the spinal fluid measured in this manner varies under different conditions, and these variations may be of considerable amount.

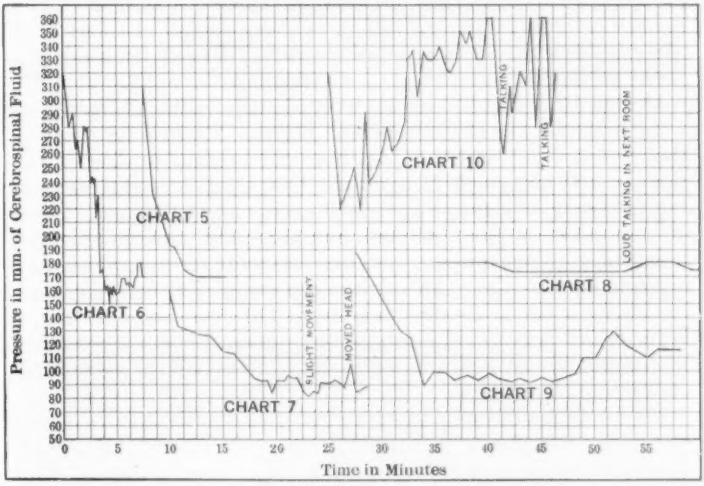



The first three charts (1, 2 and 3) indicate the effect of talking, coughing, movement of the head, pressure on the jugulars, forced respiration,[2] etc. All of these phenomena cause a considerable change in the reading of the spinal fluid manometer. The charts indicate that a change in pressure may occur. It is obvious, therefore, that one must guard against these variations in making a reading which is to represent the ordinary pressure of the cerebrospinal fluid. If the patient remains absolutely quiet and is under no great emotional strain, the reading in the manometer will reach a level at the end of five to ten minutes, which level will be maintained approximately as long as the patient is quiet.

[2] In Chart 2 it will be noted that a deep inspiration caused a rise in the pressure reading with a fall on expiration, whereas in Chart 3 a drop is recorded on inspiration and a rise on expiration, in other words a reverse is recorded on inspiration and expiration on the pressure reading in these 2 cases. Other observations have shown that in some individuals inspiration causes a rise, in others a fall. We are not clear as to the reason for this.

*This level, which is reached at the end of a period of minutes, may be considered as a standard for the cerebrospinal fluid pressure under the given conditions.*

In a vast majority of cases the reading obtained immediately after a lumbar puncture is much higher than that obtained at the end of some ten minutes. During the first portion of this ten minutes the drop is relatively great. At the end of the first three or four minutes the rate of drop becomes slower, and after seven to ten minutes a level is reached which is likely to be maintained thereafter if the patient is quiet. Although the initial drop is quite rapid, as shown in the charts, unless one is on the alert it may not be noted. The drop takes place in oscillations (see Chart 6), and the level will remain apparently stationary for several seconds,




which is about the length of time that one ordinarily observes the manometer. We wish strongly to emphasize this point, as in our own experience we were unaware of the falling level of fluid during a long period of cerebrospinal fluid pressure readings.

Out of a great many observations we present Charts 4 to 7 as illustrations of the drop that occurs. It will be noted that the preliminary drop may be very great and quite rapid, as shown in Chart 4, where the original reading was 200 mm., and at the end of three minutes the reading was 55 mm., which was maintained for two minutes until the patient began talking, when it rose to 90 mm., but again dropped to 55 mm., and then to 50 mm. after conversation ceased. Chart 5 shows a drop in the first thirty seconds from 310 mm. to 230 mm., and at the end of four minutes

a stable equilibrium was reached at 170 mm. Chart 6 shows a more irregular curve of the pressure readings. In this experiment readings were taken very frequently (*i. e.*, 13 to the minute). It shows that the drop takes place in oscillations with the level stationary for several seconds, as mentioned in the preceding paragraph. An initial reading of 320 mm. was followed by a drop to 250 mm. in the first ninety-second interval, which was succeeded by a rise to 280 mm., was unevenly sustained for nearly a minute, and then a fall to 150 mm. occurred. Chart 7 shows a case in which the drop was much slower and the fall from 160 mm. to 95 mm. or less did not occur until more than eight minutes had elapsed.

While an initial drop is the rule, it does not occur in every case, but, on the contrary, one finds at times that the original reading remains as the approximate level throughout a considerable period of observation. It is not possible, therefore, to assume that a certain percentage drop will occur in every case. In Chart 8 an observation is given in which during a period of twenty-five minutes there was hardly any change in the reading.

On the other hand, there may occasionally, although rarely, be a rise in the reading of a considerable degree. Thus, in the observation recorded in Chart 9 a drop from the original reading of 190 mm. to 100 mm. took place in the first seven or eight minutes, but at the end of eighteen minutes it again rose to 130 mm. Chart 10 represents a more marked increase. The patient was not accustomed to lumbar punctures and was quite restless, which may account for this rise. An original reading of 320 mm. was succeeded by a fall in the first minute to 220 mm., and then a rise began which is recorded in a sierra-like graph until at the end of fifteen minutes the fluid was forced out of the top of the manometer at 360 mm.

After having definitely established the observation that this drop occurs in the majority of cases, it became our interest to attempt to find an explanation of the phenomenon. The matter of local anesthetics seemed to have no appreciable effect whatsoever, as the same type of fall in pressure occurred both in the cases where the back was cocainized and in those in which it was not.

The position of the patient and the amount of emotional response seem to be important factors, of which the former takes the leading part. The usual method of performing a lumbar puncture in a horizontal position is to have the patient curl up to as great an extent as is possible, the legs flexed upon the abdomen and the head and shoulders fully flexed. Often an assistant will aid the patient in obtaining this position by placing one hand behind the knees and the other over the neck and shoulders and exerting pressure to flex the body. This position is shown in Fig. 1. It is under these conditions that the initial spinal fluid pressure reading will be found to reach its highest level and make its most marked

drop. This position puts the muscles of the patient under tension, it compresses the thorax and flexes the neck, making pressure upon the jugular vessels. All of these factors tend to cause a temporary

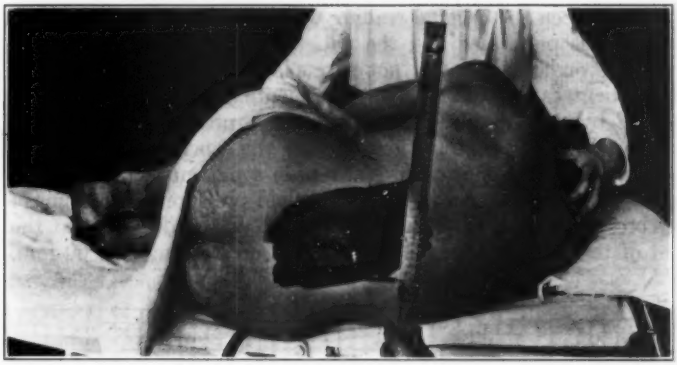


FIG. 1.—Patient flexed, knees drawn up and head bent as far forward as possible. Usual position when lumbar puncture is performed with patient in horizontal plane.

rise in the cerebrospinal fluid tension. This increase in pressure is temporary, and after a period of a few minutes "compensatory" changes occur which lead to an equilibrium.

The state of the patient's emotions with the resultant physiological effects also plays a part in the production of an increased

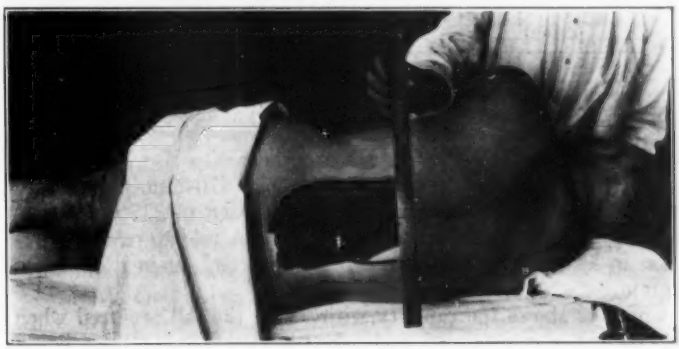


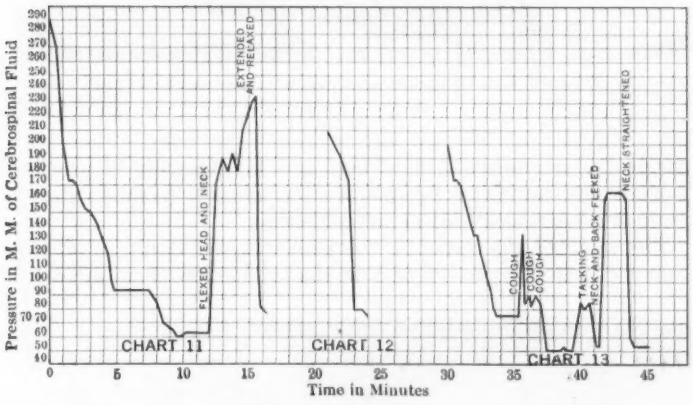
FIG. 2.—Lumbar puncture with patient extended. This position makes lumbar punctures more difficult than when patient is flexed.

pressure, as is indicated in Chart 1, showing the effect of fear. That the emotional state may play a considerable part is also indicated by the fact that injections of pituitrin and adrenalin

will cause a rise in spinal fluid pressure. Emotional changes cause increase of endocrine secretion. However, the position of the patient is a more important factor.

If the puncture is performed with the patient extended, as shown in Fig. 2, then the original reading will be much lower than if the puncture is performed and the patient in the position of Fig. 1, and a secondary drop will not take place. That the position of the patient causes the changes just mentioned has been demonstrated by us a number of times, and we present several charts which graphically portray the results of the experiments.

Chart 11 shows the readings taken on a patient who was punctured when flexed. An initial spinal fluid pressure of 290 mm. of cerebrospinal fluid was obtained. A drop occurred so that at the end of ten minutes the spinal fluid pressure reading was 60 mm.




The patient's back was then voluntarily flexed at the physician's request and the pressure immediately began to rise, and at the end of thirty seconds it reached a level of 170 mm. and at the end of three and a quarter minutes had risen to 235 mm. The patient's back was then extended and he was told to relax, whereupon in less than one minute the pressure had again dropped to 75 mm.

Chart 12 shows the rapidity with which the fall occurred when the patient was extended and relaxed, after having been flexed for the puncture.

The effect of flexion of the head and neck is well illustrated in Chart 13, which also indicates the initial drop and the effect of coughing and talking. The puncture was performed with the patient in the flexed position. At the end of seven minutes the pressure in this case had dropped from the original 190 mm. of

cerebrospinal fluid to 50 mm., at which point it remained stationary for two minutes until the patient talked, when it rose to 85 mm., remaining at this level during the period that the patient talked and on cessation of the speaking it again fell to 55 mm. During this period the patient remained flexed, but he no longer held himself rigid. The head was then voluntarily flexed to a great extent by the patient, which caused an immediate rise of 110 mm., that is, from 55 mm. to 165 mm. This was maintained for nearly a minute, when the head was again straightened, when the pressure dropped rapidly to 50 mm., where it remained; thus in this case 50 mm. would seem to be the approximate pressure when at rest.

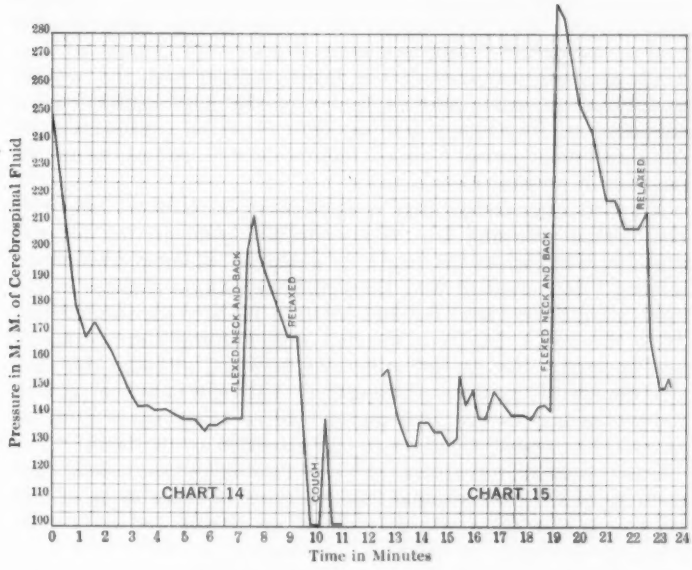



Chart 14 shows the initial drop, the rise on flexion, the secondary drop from this point which is apparently the result of the compensatory mechanism and then a further and more rapid drop that occurs upon relaxation. This experiment also shows that the relaxation and the extension of the body may cause the fall to go to a point lower than that which occurred during the preliminary drop due to compensatory mechanism alone. Even under these conditions it is not possible to be sure as to what is the real cerebrospinal fluid pressure, for after having obtained a reading that remains fairly constant for several minutes, slight changes either in position or degree of muscular tension or mental and

emotional responses may produce a further fall or a rise, as shown in Chart 9.

Chart 15 is a record of an observation in which puncture was performed on a patient accustomed to lumbar puncture. He was relatively extended and relaxed at the moment of puncture and relatively little variation in the pressure reading occurred in the subsequent six minutes. He then flexed his neck and back and the pressure reading rose in the course of a few seconds from 143 mm. to 290 mm., then slowly fell so that in about three minutes it registered 215 mm. He was then asked to relax and the reading promptly dropped to 150 mm.

It is not certain through what physiological channels these cerebrospinal fluid pressure variations, resulting from change in position, are due. The fluid pressure varies directly with the cerebral venous pressure and, to a lesser degree, with arterial tension. These are both increased in the flexed position with the muscles in contraction, while marked flexion of the neck causes jugular compression. Expectancy and fear cause increased arterial pressure. All these and probably other factors as well contribute to the increase in pressure noted.

What we have termed the "compensatory mechanism," whereby the marked rises and falls of pressure are compensated for and a stable equilibrium produced, is a matter of great theoretical interest. It is evident that through some process changes in the fluid tension are cared for and brought back to an equilibrium. After the needle has been inserted and nothing more seems to be happening, the patient will tend to relax, although he may not change his position perceptibly. This accounts for part of the preliminary drop. This matter will be taken up in a future paper. At present it suffices to call attention to the facts.

From these observations, we feel thoroughly convinced that readings of the cerebrospinal fluid pressure, in order to be of any value, must be done with exceedingly great care and must take into consideration the following conditions:

1. The patient should be relaxed both mentally and muscularly.
2. The observations must be made over a period of time of at least six or seven minutes.
3. The pressure must remain approximately constant for a period of two or three minutes before the reading can be accepted.

If these conditions are fulfilled a reading will be obtained which represents a reasonably stable pressure that exists in the patient when he is in the horizontal position and at rest. The reading obtained will depend somewhat upon whether the head is on a pillow or on the table at a level with the buttocks, whether the patient is leaning a little forward or backward, whether the needle is inserted in the median line of the dura or to the side. These factors, however, cause only a minimal variation and one that is

of no practical importance. We feel that the pressure readings obtained as outlined will serve as satisfactory standards for comparison.

These necessary conditions for approximate accuracy of spinal fluid pressure readings make it comparatively difficult to get a satisfactory reading on the average patient, unless he has been subjected to lumbar punctures previously and is fairly well trained. The average patient having his first puncture performed is fearful and tense, and he is unable to relax very completely, and is likely to fret and move if the procedure takes many minutes, as we have shown is necessary. Holding a manometer and observing it, one is apt to find five to ten minutes a long period, and unless one uses a timepiece to determine the time, he is very likely to overestimate the time that has elapsed.

When a puncture is performed, as is so often the case, upon a patient who is not coöperative, it is almost impossible to get a satisfactory pressure reading. The readings made upon patients with head trauma who are confused and resistive, or upon children who are struggling, are entirely without value because when struggling and being firmly held a reading of from 300 to 400 mm. or more may be obtained as a result of these factors, when the pressure under conditions of rest is probably not greater than 100 mm. Upon the basis of a reading of 300 or 400 mm., one might conclude that the patient was suffering from increased intracranial tension, which might not be true at all but rather a temporary rise as a result of a strained position.

Finally, we wish to call attention to the fact that in the ordinary course of human activity there are marked changes in the cerebrospinal fluid pressure. Strenuous muscular exertion must cause a temporary rise of a great extent as do various other factors. So in estimating the cerebrospinal fluid pressure one must attempt to use a standard. This standard will merely represent the pressure obtained under certain controlled conditions. The conditions that we consider absolutely requisite for obtaining workable standard of cerebrospinal fluid pressure have already been given.

**Summary.** When lumbar puncture is performed in the usual manner, with the patient lying upon his side and well flexed, the pressure recorded shortly after the needle is inserted is apt to be two, three or four times as great as that obtained after ten minutes. Usually a drop of pressure occurs, being quite rapid in the first two or three minutes, then less rapid for several minutes. Six to ten minutes after the puncture is performed, a stable equilibrium is reached and the pressure reading remains fairly constant from then on if the patient is quiet. This is not invariably true, but occurs in the majority of instances.

Several factors play a part in the production of the early high reading and subsequent fall. The most important are the position

of the patient and the amount of tension under which he holds himself. When rigidly flexed and fearful the pressure is always high. Relaxation and extension avoids this original fluid hypertension. If after the pressure has fallen the patient again makes himself rigid and flexes himself vigorously, the pressure will again rise and will fall once more. The rate of drop will be accelerated if the patient relaxes and extends himself. In order to have a standard for estimating cerebrospinal fluid pressure, it is necessary to observe the rules which are given above.

Readings taken upon patients who are fearful or struggling, as is often the case following head trauma, may be 300 or 400 per cent, or even more, in excess of the pressure existing during periods of rest and quiet.

## STUDIES OF CEREBROSPINAL FLUID IN INFANTS AND YOUNG CHILDREN IN CONDITIONS OTHER THAN ACUTE MENINGITIS.

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From October 1, 1915, to January 1, 1922, lumbar puncture was performed at the Babies' Hospital in about 800 different patients. The largest single group among this number was tuberculous meningitis; there was also a large number of cases of meningitis due to the meningococcus and a small number of other types of meningitis caused by pyogenic organisms. There were numerous cases of poliomyelitis, the majority of which occurred during the epidemic of 1916.

All the above mentioned cases have been excluded in the following study of the cerebrospinal fluid in 316 different patients. Blood-stained cerebrospinal fluids have also been excluded as well as all cases in which clinically there were any symptoms which might have been suggestive of poliomyelitis, even though paralysis was not demonstrable. The majority of the patients were under two years of age, a number were between two and three years, and a few between three and five. A brief survey of the physiology and anatomy of the cerebrospinal fluid may not be amiss:

The more modern interest in the cerebrospinal fluid dates from the important observations and masterly description of Mage die contained in his epochal monograph published in 1842.[1] Interest was further stimulated by Quincke's introduction of lumbar puncture in the living subject, which opened up a wide field for diagnostic possibilities in pathological conditions of the central nervous system.

Lying free in each ventricle are two highly convoluted vascular tufts, the choroid plexuses, the chief elaborators of cerebrospinal fluid. These project into the ventricles, carrying before them the lining epithelium. Mott[2] believes that one is justified in speaking of the plexus as the choroid gland.

The cerebrospinal fluid is contained in all the cavities of the ventricular system and in the subarachnoid space. It is probable that the fluids of these two spaces are not of common origin, although it has been proved that the major portion of the fluid is produced within the cavity of the ventricles.

Dandy[3] has proved conclusively that the fluid in the ventricles is produced by the choroid plexuses and not by the lining ependyma by the following experiment: He blocked the foramen of Monro on one side of a dog's brain, while on the other side he first removed the choroid plexus of the lateral ventricle, leaving the ependyma intact, and then blocked the foramen of Monro on that side. In the former case the ventricle became greatly distended with fluid, a condition of unilateral hydrocephalus, while in the latter it became shrunken and collapsed, and contained no fluid, although still lined by ependyma. Some portion of the cerebrospinal fluid is evidently extraventricular in origin and not simply due to a ventricular overflow through the openings in the roof of the fourth ventricle. Dandy and Blackfan[3] observed in a case of internal hydrocephalus with complete obstruction to the ventricular overflow that although only 5 cc of fluid could be withdrawn by lumbar puncture, shortly afterward a similar quantity could be withdrawn, which was obviously of extraventricular origin. Cushing and Goetsch[5] have endeavored to prove that there is an additional source of a small amount of fluid from the secretion of the posterior lobe of the pituitary gland, which is poured into the cerebrospinal fluid.

**Absorption of the Fluid.** All are familiar with the fact that cerebrospinal fluid is constantly being secreted in large quantities.[6] In fracture of the base of the skull, as well as in cases of cerebrospinal rhinorrhea, a large quantity of fluid may discharge in the course of twenty-four hours, although it is not clear whether this corresponds with the amount of fluid normally secreted during the same period.

Leonard Hill[7] has ably demonstrated that the major portion of the fluid passes into the blood. He noted that when methylene blue solution was injected into the cerebrospinal spaces the dye appeared in the bladder and stomach in from ten to twenty minutes.

The problem of how the cerebrospinal fluid finds its way to the cerebral circulation has been solved by Weed[8] by the following experiment: he showed that fluid containing granules of Prussian blue when injected into the subarachnoid space under pressure only slightly above that of the cerebrospinal fluid could be traced into villous-like processes, which project into the lumen of the sinus and eventually within the lateral walls of the sinus itself.

There is, in addition, an absorption of the fluid by way of the lymphatics (Boyd[9]), to which the fluid gains entrance by way of the perineural space which surrounds the nerves. When ferrocyanide solution is injected into the subarachnoid space the lymphatics of the neck have been found to be deeply stained. In all the spinal nerves, absorption from the perineural spaces into the lymphatics of the anterior and posterior nerve roots can be demonstrated by the ferrocyanide replacement method.

"It does, however, seem established, on definite and firm grounds, that the cerebrospinal fluid is largely produced within the cerebral ventricles by specially differentiated structures, the choroid plexuses. After traversing the ventricles, this fluid passes from the fourth ventricle into the subarachnoid spaces; these are channels adapted for fluid passage and permit the distribution of the fluid everywhere about the central nervous system. From these spaces the fluid returns to the blood-vascular system, the mode of absorption being a process largely of filtration and diffusion through arachnoid villi into the dural sinuses." (Weed.[10])

In all the examinations of cerebrospinal fluid included in this study, the cell counts were made in a counting chamber and the globulin test was done according to the method of Noguchi. For the last three years, Benedict's solution has been used to determine the presence of a reducing substance, prior to that Fehling's solution was used. In only a limited number of the cases was the pressure of the cerebrospinal fluid determined by the manometer, in the balance of the cases it was entirely a question of personal observation.

It is generally agreed that the cerebrospinal fluid of normal infants and children is clear and colorless, slightly alkaline in reaction, with a specific gravity of approximately 1002. The pressure of the fluid when the child is in the recumbent position varies between 5 and 10 mm. of mercury. There is nearly always a positive reaction for a reducing substance. Globulin does not exist in demonstrable quantities in the normal cerebrospinal fluid, and when a positive globulin reaction is present the fluid is pathological. Lymphocytes are normally the only cells present in cerebrospinal fluid. In adults up to 8 per c.mm. may be considered normal. In children they are apparently somewhat more numerous, and as many as 20 per c.mm. may be found in a normal child. None of the specimens of cerebrospinal fluid submitted in this study were examined spectroscopically.

**Normal Children.** For comparison with the data furnished by lumbar puncture in the various groups of patients with different diseases the findings in 5 normal children are presented. The cerebrospinal fluid in all the cases was clear and colorless, and the average cell count was 4 per c.mm., with a count of 8 cells as the highest. In the 4 cases in which a test for a reducing substance was

done it was positive in all. The globulin test, which was done in all cases, was positive in none.

These findings in my experience represent a normal average for infants and children. The test for reducing substance is regularly positive, and the test for globulin should be negative.

For the purpose of study and comparison with the findings of others, the cases have been classified according to clinical diagnosis. Except in the cases of congenital syphilis, lumbar puncture was generally done in all these cases because of such suspicious symptoms as convulsions, rigidity of neck and back, bulging of fontanelle, projectile vomiting, extreme irritability, changes in pulse or respiration, disturbance of the sensorium, etc.

**Respiratory Cases.** There were 67 cases in which the primary diagnosis was some form of respiratory condition other than pulmonary tuberculosis. Thirty-nine of the patients were under one year of age; most of these were cases of bronchopneumonia. There were a few cases of lobar pneumonia and a lesser number of such conditions as pleurisy, bronchitis, influenza, etc. Some of the cases were complicated by other conditions not involving the central nervous system. Of the 67 cases 21 were autopsied.

Of the entire group of respiratory cases convulsions had been noted in 15. Of the 67 cases the cerebrospinal fluid was clear and colorless in 66. In 28 cases it had been noted on the chart that it was withdrawn under increased pressure. The amounts withdrawn varied between a few cc and 35 cc. In 11 cases, 20 or more cc was withdrawn, and in 24 cases, 15 cc or more was withdrawn. The average number of cells was 11.0 per c.mm. A differential count was made in only 44 of the specimens obtained, and in nearly all of these the majority of the cells found were lymphocytes. Only 41 of the specimens of cerebrospinal fluid were tested for a reducing substance, and in 37, or 90 per cent, the test was positive. The globulin test was made in 43 of the specimens and was only positive in 5 instances, or 11 per cent. In 9 cases a routine culture of the fluid was made and in each instance it was negative. Five of the cases in this group had findings sufficiently unusual to merit further study. Of these 5 cases all had a cell count of 30 or more cells and 4 numbered more than 60 cells per c.mm.

No. 18,441, aged five months, had a generalized eczema on admission. A clear cerebrospinal fluid was withdrawn containing 90 cells, all lymphocytes, with a positive test for globulin. The infant died of bronchopneumonia, and in addition a parenchymatous degeneration of the kidneys was found at autopsy.

No. 17,513, aged fourteen months, was admitted because of cough, restlessness, and high fever. Thirty-five cc of clear fluid was withdrawn under increased pressure, in which the cell count showed 70 cells per c.mm., all lymphocytes. The child died of acute bronchopneumonia, which was noted at autopsy in addition to a fatty liver.

No. 13,561, aged seven weeks, admitted because of convulsions. The clinical diagnosis was acute bronchopneumonia; the child was discharged cured of the condition for which he was admitted. Fifteen cc of clear fluid was withdrawn under pressure with 60 cells, 99 per cent of which were lymphocytes. A reducing substance was present, but the globulin test showed no increased amount.

No. 13,084, aged six months, was a somewhat similar case; admitted on account of cough, fever and drowsiness. The leukocyte count was 31,000. The cerebrospinal fluid was clear, under increased pressure, with 62 cells per c.mm., all lymphocytes. The globulin test was positive; the test for a reducing substance was negative. The clinical diagnosis was bronchopneumonia complicated by otitis media. The infant was discharged cured of the condition for which he was admitted.

No. 11,745, was a five-year-old boy who had a lobar pneumonia with an abrupt onset; vomiting and convulsions. The cerebrospinal fluid was clear and not under increased pressure. There were 30 cells per cm., 90 per cent of which were lymphocytes. The globulin test was negative. The boy made an uneventful recovery.

Lumbar puncture was done in 3 cases of pneumonia with positive blood cultures without symptoms of meningitis. The infecting organism in 2 of these was the pneumococcus, and in the third the streptococcus. In 1 case an autopsy was done. The cerebrospinal fluid was clear and colorless in the 3 cases, and the total cells per c.mm. numbered 3, 2 and 5 cells respectively. The test for a reducing substance was positive in all, and the test for globulin was negative in each case. The above findings, together with data noted elsewhere of septicemia caused by other organisms, are interesting, as they may be viewed as possible evidence of the protective mechanism in infants afforded by the choroid plexus in delaying a blood-stream infection from invading the central nervous system.

The analysis of these findings shows that in acute respiratory infections the great majority of cases has a normal cerebrospinal fluid, while a small minority (5 of 66) shows a fluid with an increased cell count, having in a few instances an increased globulin content. The highest cell count in this group was 90 per c.mm., and practically all were lymphocytes. From the findings in the first 5 cases the diagnosis of serous meningitis might well be justified.

In 15 of the cases convulsions were present. It will be noted here as well as elsewhere in this study that in only a few of the cases was pleocytosis associated with the presence of convulsions.

**Intestinal Cases.** The following group includes the cerebrospinal fluid findings of 50 cases in which the primary diagnosis was some type of intestinal disturbance. The diseases included under this heading were: acute gastro-enteritis, acute enteritis and ileocolitis, acute intestinal indigestion and intoxication, bacillary dysentery, etc. The diagnosis was made either on discharge of the patient

from the hospital, the result of a summation of the clinical symptoms, or at autopsy, which was done in 14 of the cases. Thirty of the patients were under one year of age, and the oldest child was aged four years. The majority of the patients in this group were admitted on account of frequent stools, vomiting, loss of weight, etc. A number of complications were noted, chiefly respiratory, and acute otitis media.

Every cerebrospinal fluid examined in this group was clear and colorless. In 18 patients it was under increased pressure. Among 35 in which a test for a reducing substance was made it was found positive (normal) in 31, or 88.5 per cent. In 34 cases in which the globulin test was done it was found positive (abnormal) in only 3, or 8.8 per cent. The average cell count for 49 cases was 7.7 cells per c.mm. In nearly every instance the only cells found were lymphocytes. In 7 cases the fluid was cultured for organisms with negative results. Only 4 cerebrospinal fluids were examined in which 20 or more cells were found.

The following 4 cases are of especial interest because of the relatively high cell counts:

No. 16,521, aged twenty-seven months, admitted on account of vomiting, irritability, and anorexia, had a clear cerebrospinal fluid not under increased pressure, containing 24 cells per c.mm.; 3 of the cells were polymorphonuclear leukocytes and 21 lymphocytes. The globulin test was positive and a reducing substance was present. The child was discharged improved after the nervous symptoms had disappeared, with a diagnosis of chronic intestinal indigestion.

No. 18,564, aged four weeks, admitted on account of frequent green stools. Fifteen cc of clear fluid was withdrawn which was not under increased pressure, with 21 cells, 95 per cent of which were lymphocytes. The culture was negative and the globulin test was positive. The infant was discharged cured, with a diagnosis of acute intestinal indigestion.

No. 14,052, aged four and a half months, admitted on account of fever, cough, and labored respiration. The child was highly irritable and definite rigidity of the neck was present. The clear cerebrospinal fluid was not under increased pressure, and contained 20 cells per cm., all of which were lymphocytes. The globulin test was negative. There were no organisms present. A reducing substance was present. At autopsy acute catarrhal colitis, fatty infiltration of the liver, atelectasis, and emphysema of the lungs were found. An examination of the brain was not permitted.

No. 19,741, aged sixteen months, was admitted on account of convulsions, vomiting, drowsiness, and diarrhea. Five cc of clear cerebrospinal fluid was withdrawn which was not under increased pressure. There were 20 cells present, of which 18 were lymphocytes. No organisms were discovered. The clinical diagnosis was acute intestinal intoxication. The child was discharged cured.

In acute and chronic intestinal conditions in infancy and early childhood the great majority of cases had a normal cerebrospinal fluid, while a minority (4 of 50) showed a fluid with a slightly increased cell count, 2 having increased globulin. The highest cell count in this group was 24 per c.mm.; nearly all were lymphocytes.

The severity of the disease evidently bore no relation to the cerebrospinal fluid findings, as the analysis of the 14 cases which were autopsied indicate. In these 14 cases the highest cell count was 20 cells per cm.m., and in none was the globulin reported positive.

**Tetany.** Lumbar puncture was done in 31 cases in which the diagnosis was tetany. Twenty-six of these patients were admitted on account of convulsions, 25 were under one year of age. All but 3 were discharged from the hospital improved or unimproved of the condition for which they were admitted. In the 3 who died, autopsies were not obtainable. On the discharge records of 11 some complication was noted, the most frequent of which was rickets.

The cerebrospinal fluid in all the cases was clear and colorless, and in 15 instances was withdrawn under increased pressure in amounts which varied between 5 and 25 cc. The average number of cells per c.mm. was 7.4; 5 of the fluids had 15 or more cells. In all but a few of the specimens of fluid the only cells were lymphocytes. In 20 cases in which a test for globulin was done it was found positive in 1 case and slightly positive in another. In 6 cases in which the cerebrospinal fluid was cultured all were reported negative.

Of especial interest are the following cases because of their atypical cerebrospinal fluid findings:

No. 19,238, aged three months, admitted on account of vomiting, convulsions, and rigidity of neck. The infant had an exceedingly well-marked Chvostek reaction. Five cc of clear fluid was withdrawn with 25 cells per c.mm., all of which were lymphocytes. The Pirquet test was positive. The child was discharged from the hospital improved of the condition for which he was admitted, and was not seen subsequently.

No. 19,434, aged twenty months, was admitted on account of fever, irritability, and convulsions. Five cc of clear fluid was withdrawn not under increased pressure, with 30 cells per cmm. The Chvostek reaction was marked. The child was discharged improved with a diagnosis of tetany complicated by nasopharyngitis.

An interesting observation regarding the patients with tetany was the low average cell count in a group of cases in which all but 5 had convulsions. This is fairly conclusive evidence that even repeated convulsions bear no relation to the number of cells in the cerebrospinal fluid of infants and very young children.

In all the cases of tetany the cerebrospinal fluid was essentially normal; in a small number only (5 of 31) showed a fluid with a slightly increased cell count.

**Pyelitis.** Lumbar puncture was done in 6 infants acutely ill with pyuria, all of whom were under one year of age. Four of these were discharged cured from the hospital, with a diagnosis of pyelitis. Two died and came to autopsy. In 1 pyelonephritis, bronchopneumonia, and follicular colitis were found, and in the other parenchymatous degeneration of the kidneys, fatty liver, and a general visceral congestion. Two of these infants were admitted because of convulsions, the others because of drowsiness, vomiting, fever, etc. The cerebrospinal fluid was clear and colorless in every patient, and in no instance was it under increased pressure. The average count for the 6 cases was 12 cells per c. mm., and in only 1 case were there as many as 20 cells. This was noted in an infant, aged eleven months, who died of pyelonephritis with uremia. There was a reducing substance present in 5 of the specimens of cerebrospinal fluid, of which only that number were tested. The globulin test was negative in all of the 6 cases.

The cerebrospinal fluid was uniformly normal in 6 infants acutely ill with pyuria. The cerebrospinal fluid was clear and colorless with a reducing substance present, no increase in globulin, and having an increased number of cells in but 1 case.

**Neuropathic Children.** Four children in whom lumbar puncture was done were discharged with the notation that they were "neuropathic children" in whom no disease was established. The youngest of the 4 was aged twenty-one months, and the oldest three and a half years. They were admitted on account of symptoms suggesting tuberculous meningitis. Two were discharged cured and 2 improved of the symptoms for which they were admitted. The cerebrospinal fluid in the 4 cases was clear and colorless and in only 1 instance was it under increased pressure. The cell counts were 2, 10, 15, and 30 cells respectively. Two of the specimens of cerebrospinal fluid had negative cultures which included the case showing 30 cells per c.mm., and in the others cultures were not made. The globulin was negative in all 4 cases, and the test for a reducing substance was positive in every case. That is, all 4 had normal cerebrospinal fluids, 1 of which showed an increased number of cells.

**Otitis Media.** Lumbar puncture was done in 8 cases in which the only disease noted was acute otitis media. In 4 of the cases the disease was bilateral. The cerebrospinal fluid findings are presented in these cases as evidence against the belief held by some clinicians that there may be an increased cell count in acute middle ear infections. Otitis media is a frequent complication among patients tabulated in the entire study. It might be questioned whether the complicating middle ear infection might not have been the cause of some of the unusual cell counts in certain of the cases rather than the primary condition of pneumonia, etc. The normal cerebrospinal fluids which were found when the otitis was the only disease present proves rather conclusively that otitis in itself exerts no change in

the cerebrospinal fluid. Nor is there any reason why this should not be so. The average age of the 8 cases was ten months. In every instance the fluid was clear and colorless with the pressure increased in 3. The average number of cells per c.mm. was 5.6 and the highest count only 10 cells. Among 4 specimens tested for a reducing substance, 3 were positive. The globulin test was positive only once in the 8 cases.

The cerebrospinal fluid of 8 cases of acute otitis media was essentially normal in 7 cases with no increase of cells in any of the cases. In 1 case with a clear cerebrospinal fluid containing 10 polymorphonuclear leukocytes per c.mm., globulin was present in sufficient amount to cause a reaction.

**Cerebral Thrombosis.** Lumbar punctures were done in 5 cases of cerebral thrombosis proved at autopsy, 3 of which were due to sepsis and 2 to marasmus. The patients were aged fourteen months, sixteen months, nineteen days, seven and a half months, and seventeen months, respectively. In 2 cases the cerebrospinal fluid was cloudy yellow in color; the balance were clear and colorless.

No. 16,265, a septic case of thrombosis, was admitted because of vomiting and twitching of the right side of the body. Twenty cc of cerebrospinal fluid under normal pressure was removed. It contained 32 cells, of which 60 per cent were polymorphonuclear leukocytes and 40 per cent were lymphocytes. No film was present and no organisms were discovered. A reducing substance was present. At autopsy a thrombosis of the lateral and longitudinal sinus was found in addition to mastoiditis and cerebral hemorrhage.

No. 16,060, a case of marantic thrombosis, aged sixteen months, was admitted because of stupor. The leukocyte count was 40,000. Seventy-five cc of clear cerebrospinal fluid was withdrawn under greatly increased pressure. Only 5 cells were found, 3 of which were lymphocytes. The globulin test was negative; the test for a reducing substance was positive. There were no organisms found. At autopsy, cerebral thrombosis and hemorrhage were present, in addition to otitis media and congenital cardiac anomaly.

No. 18,580, a case of marantic thrombosis, aged nineteen days, admitted on account of dyspnea and rigidity of neck. Ten cc of yellow cloudy fluid under normal pressure was withdrawn which contained 46 cells, of which 25 per cent were lymphocytes and 75 per cent polymorphonuclears. The test for globulin was positive. At autopsy multiple cerebral thrombi were found as well as multiple cerebral hemorrhages. The yellow color is thus explained.

No. 16,873, a case of septic thrombosis, aged seven and a half months, was admitted because of enlargement of the head, stupor, and vomiting. The fontanelle was tense and the knee jerks exaggerated. The cerebrospinal fluid was clear and under normal pressure; 2 cells were found both of which were lymphocytes. A reducing substance was present, the globulin test was negative. Cerebral

thrombosis and internal hydrocephalus were present at autopsy, in addition to osteomyelitis of the malar bone. The hydrocephalus is difficult to explain with 2 cells only per c.mm., and a negative globulin test.

No. 20,690, aged twenty-nine and a half months. The cerebral spinal fluid was yellow tinged, faintly turbid, with a normal pressure, 8 lymphocytes, and 530 crenated red blood cells were found. The globulin test was positive, a reducing substance was present, and the culture negative.

At autopsy, thrombosis of cerebral sinuses and veins, cerebral softening, acute leptomeningitis, and hypostatic pulmonary congestion were present.

In only 1 of the 5 cases was the cerebrospinal fluid under increased pressure. The average cell count for the 5 cases was 18 cells per c.mm. In 2 of the cases the polymorphonuclear leukocytes predominated. The reaction for a reducing substance was positive in 4 cases in which the test was done, and the globulin test was positive in 2 of 4 tests. It would appear from the findings in the limited number of cases here presented that there is nothing pathognomonic in the cerebrospinal fluid of cerebral thrombosis. It is significant that 2 of the cases showed a yellow colored fluid, apparently the result of a hemorrhage following thrombosis.

**Congenital Syphilis.** Lumbar puncture was done in 20 cases of congenital syphilis. In 8 the diagnosis was proved at autopsy and in the others either by florid clinical manifestations or by the Wassermann test, or both. The average age of the patients in this group was ten and six-tenths months; 12 were less than six months of age. In former years lumbar puncture was done in congenital syphilitic cases only when an invasion of the meninges was suspected. More recently it has been done somewhat routinely in such cases.

In every case but 2 the cerebrospinal fluid was clear and colorless, and in only 6 instances was increased pressure noted. From 5 to 50 cc of fluid was withdrawn. Excluding the 3 cases proved at autopsy to be syphilitic meningitis, the average cell count for the remaining 17 cases was 15.5 per c.mm. Lymphocytes predominated in all the differential counts of the cells and in most instances no polymorphonuclears were found. A test for a reducing substance was done in only 11 cases, and in 8 was positive.

The globulin test which was done in all the cases was positive in 9, faintly positive in 1, and in the remaining 10 negative. Although the average cell count in the cases of congenital syphilis runs slightly higher than the number generally considered normal, there is great variability in the different cases. The specimens of fluid with the highest counts (with the exception of one of the cases with cerebral lues) occurred in infants in the first few months of life. Especially interesting are the low cell counts (5–7–5) in 3 cases with a positive Wassermann reaction of the cerebrospinal

fluid, which suggests that congenital syphilis may involve the central nervous system without increase in the number of cells in the cerebrospinal fluid.

The intensity of the demonstrable syphilitic manifestations apparently may have no relation to the number of cells in the cerebrospinal fluid, as case No. 17,781 illustrates. This was a three weeks old infant who had, in addition to the common syphilitic manifestations, general anasarca. Five cc of clear cerebrospinal fluid was withdrawn under normal pressure. Only 3 cells were found, and the globulin test was negative. The autopsy diagnosis were congenital syphilis with acute bronchopneumonia, chronic interstitial splenitis, acute splenic tumor, and degeneration of the kidneys. An examination of the head was not permitted.

No. 20,219 is an example of a child with severe manifestations of infection and a high cell count of the spinal fluid. This occurred in a nine weeks old infant admitted on account of general anasarca in addition to cutaneous syphilitic manifestations. The cerebrospinal fluid was clear and colorless and contained 44 cells, all of which were lymphocytes. There was a reducing substance present, and the globulin test was negative. The autopsy diagnosis was congenital syphilis with chronic interstitial splenitis, chronic perisplenitis, chronic interstitial hepatitis, acute diffuse nephritis, and general anasarca. Permission to examine the head was not obtainable.

The cerebrospinal fluid findings in the 3 following cases of syphilitic meningitis (proved at autopsy) are important:

No. 11,897, aged one year, with a luetic history on the paternal side and a mother who gave a history of 2 stillbirths and 2 infants who died directly after birth; all premature. The child was admitted because of vomiting, increasing drowsiness, and retraction of the head. The infant died four days after admission. Fifteen cc of slightly turbid cerebrospinal fluid was withdrawn under moderately increased pressure, with 650 cells per c.mm., 66 per cent of which were lymphocytes. The globulin was moderately increased. The cerebrospinal fluid of 3 lumbar punctures showed little variation. The cultures were negative; no tubercle bacilli were present. Autopsy showed congestion of the meninges and edema of the pia. Small, thickened grayish-white patches were noted over the pia mater in the sulci of the parietal and temporal lobes. There was considerable softening of the brain. Hemorrhages and minute thrombi in the gray matter were found. A moderate hydrocephalus was present.

No. 19,042, aged four months, was admitted because of drowsiness and irritability of two days' duration. The parents were both luetic. The infant died two days after admission. Eight cc of semi-turbid cerebrospinal fluid was withdrawn by lumbar puncture under increased pressure, with 810 cells per c.mm., 90 per cent of which

were lymphocytes and the balance polymorphonuclears. The globulin test was positive. No organisms were found. At autopsy it was found that the vessels of the pia were uniformly congested over all the surfaces of the cerebrum and cerebellum. The pia was slightly cloudy and thickened, especially over the interpeduncular space at the base.

No. 12,092, aged nine weeks, admitted because of convulsions of twelve days' duration and cyanotic attacks dating from birth. The child died two weeks after admission to the hospital. Eight cc of clear and colorless fluid was withdrawn by lumbar puncture under normal pressure. The test for globulin was negative with both the Noguchi and the ammonium sulphate methods; the test for a reducing substance was negative; 8 cells per c.mm. were found, of which 99 per cent were lymphocytes. No tubercle bacilli were present. The autopsy showed congestion of the pia over both sides of the cerebrum and a turbid pia over the base of the brain. In addition to the syphilitic meningitis, perisplenitis, parenchymatous degeneration of kidneys, and asymmetrical microgyria were noted.

The cerebrospinal fluid in congenital syphilis is clear and colorless (excepting in certain cases of syphilitic meningitis), and usually under normal pressure. The cytology is a variable factor. There is evidence in this series of cases to demonstrate that there may be involvement of the central nervous system without pleocytosis. The intensity of the clinical manifestations is apparently not a criterion to the cytology.

**Brain Tumor.** Twenty-one patients on whom lumbar puncture were done were diagnosed clinically as brain tumor. In 8 cases the diagnosis was proved at autopsy; in another it was proved by the subsequent course as observed in another hospital. The average age of the children in this group was approximately three years. Most of these children were admitted on account of such symptoms as vomiting, headache, disturbance of vision, abnormal gait, and convulsions.

In at least 11 of the patients the tumor was so far advanced that the diagnosis was made on admission to the hospital. In 6 of the cases which came to autopsy the tumor was found to be a glioma, in the other 2 the type was not recorded. In 2 of the cases the clinical diagnosis was tuberculoma, and in the balance of the patients it was stated to be cerebral or cerebellar tumor. The cerebrospinal fluid was clear and colorless in all but 2 of the cases; in 1 of these it was yellow and in the other straw colored. In only 13 of the cases was the fluid withdrawn under increased pressure.

Amounts varying between 2 and 3 cc were withdrawn by lumbar puncture. The average cell count was 52.2 cells per c.mm. A differential count was made in only 9 of the cases, and in 7 of these there was a great preponderance of lymphocytes. Both of the counts with a preponderance of polymorphonuclears were in cases which

came to autopsy. One was a cerebral gliosarcoma with hydrocephalus, and the other a neoplasm of the cerebrum with a chronic pachymeningitis. Although there was a high average cell count for the group of brain tumor cases, caused by a few relatively high counts, the general average for the balance was low, as there were 13 counts which were not higher than 15 cells per c.mm. Among tests made for a reducing substance in 16 cases 12 were positive. Of 16 globulin tests only 6 were positive. Of 7 Wassermann tests all were negative.

Two cases have been selected, both proved by autopsy, which illustrate the variable fluid findings both as to cell count, globulin, and reducing substances.

No. 13,855, aged ten months, admitted on account of vomiting of three weeks' duration. The fontanelle was tense and bulging, the knee jerks were exaggerated; the infant was quite drowsy. The leukocyte count was 13,000 and the temperature normal. Twenty cc of clear fluid was withdrawn under pressure, which contained 90 cells per c.mm., of which 91 per cent were lymphocytes and 9 per cent polymorphonuclear leukocytes. There was no reducing substance present, the Wassermann of the spinal fluid was negative, and the globulin test positive. At autopsy a cerebellar glioma was found which had caused internal hydrocephalus.

No. 20,035, aged thirteen months, was admitted on account of progressive loss of weight and vomiting, which began five months prior to admission. Fifteen cc of clear fluid, not under increased pressure, was withdrawn by lumbar puncture. The fluid contained only 13 cells per c.mm., of which 12 were lymphocytes and 1 a polymorphonuclear leukocyte. There was no film. The test for a reducing substance was positive, and the globulin negative. At autopsy a subpial cerebellar tumor was found, bronchopneumonia, acute mediastinitis, and retropharyngeal abscess.

All the cases of brain tumor but 2 had cerebrospinal fluid that was clear and colorless, and in only 13 of the 21 cases was the fluid withdrawn under increased pressure. In 11 of the 21 cases the cell count was not more than 10 per c.mm. The average for 20 cases was 52.2 per c.mm. and the highest was 240. Among 16 tested for a reducing substance all but 4 were positive. In 15 tested for globulin only 6 were positive. One is impressed by the fact that the cerebrospinal fluid in the cases of cerebral neoplasm presented in this study shows nothing pathognomonic.

**Tuberculosis.** Lumbar puncture was done in 7 cases of tuberculosis without symptoms of tuberculous meningitis, 6 of which were cases of acute general miliary tuberculosis (3 proved at autopsy) and 1 of pulmonary tuberculosis with a complicating corneal ulcer. All of the patients were under one year of age but 1, who was aged four years. The specimens of cerebrospinal fluid were clear and colorless in every case, and the average cell count for 6 cases was 12.5

cells per c.mm. Of 5 cerebrospinal fluids tested for a reducing substance all were positive. Differential counts of the cells were done in only 4 cases; 3 of these showed a great preponderance of polymorphonuclear leukocytes, which might possibly be explained as a reaction to beginning invasion of the meninges. The diagnosis of tuberculous meningitis is made at the Babies' Hospital only when tubercle bacilli are found in the spinal fluid.

Case 17,718 is of interest, as it demonstrates that an extensive pulmonary and miliary tuberculosis may exist without an invasion of the meninges by the tubercle bacilli. The patient was twelve months of age, admitted on account of cough, fever, and somnolence. The right ear was discharging and, as a right facial paralysis was present, this was probably a tuberculous otitis. Ten cc of clear cerebrospinal fluid under normal pressure was withdrawn with 12 cells per c.mm., 80 per cent of which were lymphocytes. The globulin test was positive and a reducing substance was present. Three lumbar punctures were done in an effort to find tubercle bacilli, but none were demonstrable. At autopsy general miliary tuberculosis was found in addition to tuberculosis of the lungs. Permission to open the head was not obtainable. The presence of 90 per cent of polymorphonuclears in Case 11,925 is difficult to explain. The patient was ten months of age and admitted on account of rigidity of the neck and back. Fifteen cc of clear cerebrospinal fluid was withdrawn under pressure with 32 cells per c.mm., 90 per cent of which were polymorphonuclear leukocytes. No tubercle bacilli were found; the test for a reducing substance and the globulin test were positive. At autopsy miliary tuberculosis of the lungs, spleen, liver and lymph nodes was found. Permission to open the head was not obtainable.

The analysis of the few cases of general miliary tuberculosis show that the cerebrospinal fluid is essentially normal, always clear and colorless with a reducing substance always present. Once only in 6 cases was an increased cell count noted, and in 2 of 3 cases autopsied the test for globulin was positive.

**Status Lymphaticus.** Lumbar puncture was done in 4 cases which at autopsy were found to be status lymphaticus. The patients were aged twenty-eight months, two and a half months, three months, and nine weeks, respectively. Two were admitted because of convulsions and the others for symptoms suggestive of meningitis. In the 4 cases the cerebrospinal fluid was clear and colorless, and in 3 was withdrawn under increased pressure. In 3 of the specimens the cell count was 12, and in 1 it was 10. In 3 there was a reducing substance present; in all 4 the globulin test was negative. These few cases are not included with the idea that any conclusions might be drawn from such a limited number, but that they might form a nucleus to which could be added cases of other observers.

It may be noted that the cerebrospinal fluid was essentially

negative in 3 of the cases. The details of the case with abnormal cerebrospinal fluid are as follows: No. 17,924, aged twenty-eight months, was admitted because of high fever (temperature 105.4 F.), persistent vomiting and diarrhea. The child was highly irritable; the knee jerks were exaggerated; there was no rigidity of the neck. The leukocytes numbered 42,000. The cerebrospinal fluid was withdrawn under increased pressure and showed 12 cells per c.mm., 75 per cent of which were polymorphonuclear leukocytes. The reaction for a reducing substance and globulin was negative. A culture of the cerebrospinal fluid was sterile. Status lymphaticus, fatty liver, and subpleural hemorrhages were found at autopsy. The preponderance of polymorphonuclear leukocytes may possibly be explained by an early invasion of the meninges, which might have developed into pyogenic meningitis had not death been caused by the associated status lymphaticus. This explanation does not go well with a sterile cerebrospinal fluid.

**Summary.** In acute respiratory infections the cerebrospinal fluid is usually normal. In certain cases which had an uninterrupted convalescence without sequelæ the cerebrospinal fluid showed a pleocytosis with or without a positive globulin reaction during the acute stage of the disease. It is believed that in certain of these cases the diagnosis of serous meningitis might be entertained. In 15 of these cases of pneumonia convulsions were present. In many of the cases from the other groups convulsions were also noted, yet in only a few was a pleocytosis associated with the presence of convulsions.

In acute and chronic intestinal conditions in infancy and early childhood the great majority of cases have a normal cerebrospinal fluid while a minority show a fluid with a slightly increased cell count.

In all the cases of tetany the cerebrospinal fluid was essentially normal; a small number only (5 of 31) showed a fluid with an average cell count of 21. An important observation regarding the patients with tetany was the low average cell count in a group of cases in which all but 5 had convulsions. This is fairly conclusive evidence that even repeated convulsions, which were observed in many of these cases, bear no relation to the number of cells in the cerebrospinal fluid.

In a group of 4 "neuropathic children" all had normal cerebrospinal fluids. In 1 of these the number of cells reached the upper normal limit.

Evidence is offered that there is no increased cell count in cases of acute otitis media by the findings in 8 cases with an average of 5.6 cells per c.mm.

It would appear from the variable findings in 4 cases of cerebral thrombosis that there is nothing pathognomonic in the cerebrospinal fluid.

The cerebrospinal fluid in congenital syphilis is clear and colorless (excepting in certain cases of syphilitic meningitis), and usually under normal pressure. The cytology is a variable factor. There is some evidence in this series of cases to demonstrate that there may be involvement of the central nervous system without a pleocytosis. The intensity of the clinical manifestations is apparently not a criterion to the cytology.

In cases of brain tumor one is impressed by the variable cerebrospinal fluid findings. In 11 of 21 cases the count was not higher than 10 cells per c.cm. In 7 cases the count was more than 100. In 15 cases tested for globulin only 6 were positive.

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## THE VALUE OF CRANIAL DECOMPRESSIVE OPERATIONS.

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This communication summarizes the observed effects of the various forms of cranial decompression as determined by a study of the clinical material admitted to, and operated upon in the surgical service of Mt. Sinai Hospital since 1915. The indications for this general form of operation have included tumor of the brain (approximately 78 per cent) and cranio-cerebral injury (approximately 11 per cent). Eighty-nine cases were studied. The general statistical information is as follows:

| | Cases. |
|---|---|
| Decompression in all types of cases with and without preceding exploration or other operative procedure | 89 |
| Died | 45 |
| Decompression for brain tumor with and without preceding exploration or other operative procedure | 70 |
| Died | 29 |
| Decompression for cranio-cerebral injury | 10 |
| Died | 5 |
| Decompression for disease, malformation, etc. | 9 |
| Died | 5 |
| Subtemporal decompression with and without preceding exploration or other operative procedure | 41 |
| Died | 22 |
| | |
| Subtemporal decompression | 26 |
| Died | 13 |
| Bilateral subtemporal decompression | 3 |
| Died | 1 |
| Subtemporal decompression plus other operative procedure such as contralateral osteoplastic flap, etc. | 15 |
| Died | 9 |
| Suboccipital operations; decompression[1] always following exploration | 27 |
| Died | 13 |

The patients in this series generally presented a comparatively poor operative risk. This was due to a deterioration of the general resisting powers of the individual produced primarily by the presence of a long standing intracranial lesion; and secondarily by the association of some other disease, notably, cardiovascular disease. The operations done upon these patients were performed by a number of different surgeons at the hospital.

The mortality following the operation is naturally in direct proportion to the gravity of the indication. In a small number of cases of inflammatory and degenerative disease, in whom the decompression was done under error of diagnosis, the mortality was highest. In cranio-cerebral injuries the gravity of the injury determines the mortality figure absolutely; and inasmuch as decompressive operations are not done for injury except when the latter, or its effects, is most severe, the mortality is, naturally, very high. Irremovable or unlocalizable tumors of the brain showed the lowest general immediate mortality after decompression.

The mortality of cranial operations is, to a large degree, dependent upon the extent of the procedure and the amount of intracranial manipulation. The highest mortality occurred when the decompression was added to some other procedure, such as an osteoplastic flap on the contralateral side. In these patients the decompression was often added because no definite site of disease was demonstrable in the exploration and it was hoped that the added decompression would relieve the symptoms of increased intracranial pressure of brain tumor or cranio-cerebral injury. These were all, naturally, subtemporal operations.

[1] It is questionable how much decompression results from this operation.

In this series, the subtemporal operation failed more often to relieve symptoms or to prevent a fatal outcome than the suboccipital operation: The difference is due entirely to the fact that the risks of the subtemporal operation were more willingly assumed in far advanced cases of supratentorial disease than were those of the suboccipital operation in equally far advanced conditions in the subtentorial fossa.

The subjoined table[1] demonstrates that these cranial operations are better borne in the earlier periods of life. The table includes all types of decompressions practised either alone or added to a major cranial operation.

| | Patients. | Mortality, per cent. |
|---|---|---|
| From 1 to 5 years | 5 | 25 |
| 5 to 10 " | 7 | 28 |
| 11 to 20 " | 9 | 44 |
| 21 to 30 " | 21 | 13 |
| 31 to 40 " | 9 | 44 |
| 41 to 50 " | 17 | 64 |
| 51 to 60 " | 4 | 75 |

In the brain tumor cases the length of time for which the lesion was present prior to operation seems to make little difference in the mortality provided the general condition of the patient is equally good. It is noteworthy that a proportionally larger mortality was present in the patients in whom the manifestations of neoplastic disease were present a relatively short time—a few weeks. The explanation for this state of affairs lies in the fact that such patients surely had the tumor growing for a much more considerable time without the exhibition of any subjective symptoms. Then some striking and dominating phenomenon occurred, such as coma, or a hemiplegia, or one or more convulsions, which brought the subject forcibly to the patient's attention at a time when the disease was relatively far advanced; at this late time any operation added but another overwhelming insult when the resisting powers of the individual were at a minimum or were entirely gone. In the earlier years the tendency to interfere in such patients was much stronger than now; since then the futility of operating in such cases is recognized. The pathological basis for this lies in the occurrence of some accident in the tumor, especially hemorrhage, leading to pressure upon some vital area.

The cause of death after decompressive operations performed for cranio-cerebral injury has always been the effects of a very grave injury possibly plus the shock of the operation; death always took place within a few hours. The primary cause of death after decompression when done for brain tumor is always a long continued grave intracranial lesion. In the cases in which some other

[1] The differences are not quite as marked as the figures would indicate because, as age increases, other complicating factors enter which must be taken into account.

intracranial procedure preceded the decompression, the former was a most important contributory cause. Puncture of the brain in infiltrating tumors deserves especial mention as in about 20 per cent of the cases in which it is done, death follows in from one one to two weeks from hemorrhage into the tumor.

Decompressions are of two kinds. In the one a temporary effect is wanted for the purpose of permitting some other procedure. In the other a more lasting effect is desired for the purpose of relieving some or all of the symptoms of tumor of the brain. A decompressive effect upon the brain can be produced in a variety of ways. In infants and young children with hydrocephalus resulting from obstruction in the aqueduct or fourth ventricle, in whom the fontanelles are still open, decompression can be accomplished by aspirating the ventricle directly through the space unguarded by bone. The opportunities for this procedure were, however, uncommon. Unfortunately, too, the effect is very temporary and the procedure must be repeated. An attempt was made to obviate the disability of repeated aspiration when the corpus callosum puncture was devised; while the effect of the latter is more lasting in the successful cases than simple aspiration, it, too, has a relatively short period of usefulness in many cases.

In doing an exploratory operation upon the brain, in which a cranioplastic flap is turned down, decompression can be effected by removing the bony part of the flap. In previous years, this was sometimes unavoidable owing to an excessive bulging of the intracranial contents; but with modern methods of diminishing intracranial pressure, the need for this is relatively rare. The deformity resulting from such an operation is a progressive one, becomes very marked and the underlying brain is unduly exposed to all kinds of traumata; so that this makeshift procedure in the fronto-parieto-occipital regions is very undesirable and is practically never done now if one can help oneself otherwise. This series includes no case of this kind. A somewhat similar, though, to be sure, a much less marked effect can be accomplished by biting away a small segment at one of the lower angles of the bone flap and leaving the underlying dural flap partially open; most operators do this. When well planned and executed there is in many cases very little deformity and the brain is not exposed to injury. For many years, there has been a tendency to employ this technic in some of the brain cases for decompressive purposes.

Ordinarily, however, the operations practised include only the standard forms of the subtemporal (Cushing) and the suboccipital operation. In the earlier years attempts were made in a few of the cases to enhance the effects of the decompression—most commonly the subtemporal variety—by doing in addition a corpus callosum puncture. In this series the results were poor, however, and this additional procedure has not recently been done.

The subtemporal operation is always done on the right side in left handed persons; ordinarily it is a unilateral operation; but in several patients a bilateral operation was done either in one or two sittings. The technic includes the following steps: (1) Usually a bow-shaped incision is made over the temporal fossa planned and executed to avoid the temporal artery and the frontal and orbicular branches of the facial nerve; in rare instances a vertical incision is made; (2) division of the temporal fascia and muscle in a vertical direction adequately to expose the entire temporal fossa; (3) opening of the skull with a hand trephine and the enlargement of the opening by undercutting with appropriate forceps so that it measures finally 5 by 7 cm. as a minimum; (4) opening of the dura by a crucial incision and leaving it wide open at the conclusion of the operation; (5) careful suture of the outer wound.

In the suboccipital operation the usual cross-bow incision is made. The soft parts and muscles are pared from the bone and the subtentorial fossa is opened into on either side of the mid-line with a hand trephine. Enough bone is removed with rongeur forceps to adequately expose the cerebellum. This includes all of the flat surface of the occipital bone lying between the grooves for the lateral sinuses and the margin of the foramen magnum; the margin of the latter is always excised as soon as possible in order to prevent any sudden strangulation of the medulla. Latterly, the bone is removed well into the mastoid; the limit of this extension is the site of emergence of the stylomastoid artery from its foramen. The dura is opened from side to side across the median line and vertical incisions are added at either side up to the transverse sinus.

Decompression in Brain Tumor. A certain amount of exploration is often done in the general area of the operative field. Subtemporal operations are done purposely as decompressive procedures; suboccipital operations always begin as explorations and when no disease is demonstrable, the results of the procedure serve although not done with this object in mind, for decompressive purposes. When there is very great increase in intracranial pressure and the brain substance prolapses into the opening of the dura, exploration is not possible. With modern technic attempts are always made to prevent this by emptying the ventricles as a preliminary measure; not always is this, however, successful both because the ventricles may contain only an insufficient amount of fluid, or, in rare instances, because the exploring needle escapes entering the ventricle. When much fluid can be removed from the ventricles, or, in certain cases, from large cystic collections due to the breaking down of the tumor, the manipulations are rendered much more simple and a fair amount of exploration can be accomplished; a greater degree of decompression is also assured in such cases. In the cerebellar cases emptying of the cisterna

magna is a great additional aid. Although our experience is small we have seen no beneficial decompressive effect resulting from the administration of concentrated solutions of sodium chloride.

In the earlier years the operations were practically always done under general anesthesia. It has latterly been recognized that this is a distinct disadvantage: The anesthetic drug increases the intracranial tension and adds more difficulty and danger to the intracranial manipulations; anesthesia carries with it, among other things, the dangers of postoperative pneumonia. So that, more and more, resort is being had to local anesthesia. As a general rule the patients tolerate this form of analgesia remarkably well and the operations can be satisfactorily done.

In the subtemporal cases the amount of shock or other appreciable effect is at a minimum. In the suboccipital cases the effect is much more pronounced. The fall of blood-pressure is sometimes considerable even in patients who are in very good general condition before the beginning of the operation; sometimes the pressure falls to 60 or 70 mm. Hg. by the time the dura is exposed; this happens in the absence of any loss of blood and is apparently explainable by the relatively larger extent of the surgical procedure and by the difference in location of the site of operation and its proximity to the vital medullary centers. The much longer time necessary for the performance of the suboccipital operation, as opposed to that necessary for the subtemporal operation, and the proportionally greater exhibition of anesthetic, when the latter is employed, is a powerful contributory factor in the production of shock in the suboccipital operation.

The manipulations necessary for the performance of either the subtemporal or suboccipital operation, in cases in which there already preëxists considerable increase of intracranial pressure and bulging of the brain, are ready causes for the production of variable degrees of edema of the brain. So that sometimes the patients' general condition shows some deterioration and individual symptoms show some increase in the first twenty-four or forty-eight hours after operation. Thereafter as the edema disappears and the static conditions in the cranium become accustomed to the new changes, the symptoms subside and improvement begins to be perceptible in those cases which are favorably influenced by the operation.

In a few of the cerebellar cases it was noted that following the operation a variable degree of rigidity of the neck was present in the first twenty-four or forty-eight hours after operation. It seems reasonable to assume that the latter was due either to the operative trauma in the musculature of the neck and the early stages of the consequent reparative inflammation necessary to the healing of the wound, or to some extravasation of blood. Somewhat similar phenomena to the last are present in cases of fracture

of the base of the skull; these are due to irritation of the meninges with blood; they were referred to in a previous communication.

There has been no case of brain tumor in this series in which there has been a complete disappearance of all of the elicitable neurological disturbances following a decompressive operation. There has been no case of brain tumor in this series in which, following the decompression, the relief of the subjective symptoms continued for a sufficiently long time to warrant the opinion that the lesion existing in the brain had remained stationary because of the operation. For these opinions it must be remembered that some of the neoplastic lesions are of extremely slow growth and that the palliation may continue unbroken for a number of years. From the point of view of the patient, palliation is sought for certain very perceptible and incapacitating disturbances, such as headache, disturbance of vision or speech, convulsions, paralyses or muscle weaknesses, and when such gross disabilities become lessened or disappear after operation, and the disappearance becomes noteworthy by its apparent persistence, it is assumed by the patient that a cure has been attained. From the point of view of the neurologist the assumption may not be justified because the previously existing abnormal neurological phenomena may continue to show progress, or new disturbances of neurological function may appear and grow more pronounced which indicate only too well to the neurologist that more and more brain tissue is becoming involved by the growing tumor.

Generally speaking 43 per cent of the cases of brain tumor subjected to decompression and in whom the operation was successfully carried out, were not relieved by the operation and left the hospital in an unchanged physical condition. Twenty-four per cent were only slightly benefited; 20 per cent were moderately benefited; 14 per cent were markedly benefited. After the subtemporal operation 23 per cent showed no change; 14 per cent showed only a slight improvement; 20 per cent a moderate improvement; and 7 per cent a marked improvement. After the suboccipital operation 10 per cent were slightly improved and 7 per cent were markedly improved. These figures are classified in the table for clarity:

| | Subtemporal decompressions, per cent. | Suboccipital operations, per cent. | Total per cent. |
|---|---|---|---|
| Unchanged | 23 | 20 | 43 |
| Slight relief | 14 | 10 | 24 |
| Moderate relief | 20 | .. | 20 |
| Marked relief | 7 | 7 | 14 |

The determination of these percentage figures is based upon the general manifestations of brain and such outstanding subjective and objective symptoms as were indicated previously (headache, vision, convulsions, etc.). The proper comparison of the relative

importance of the various symptoms, their proper evaluation as compared with one another and from one case to the other, and their proper integration has been especially difficult. It is believed, however, that the figures are essentially correct and reflect the true state of affairs.

The differences in the various grades of relief which decompression has afforded have been more marked in cases of supratentorial neoplastic disease and after the subtemporal operation than with subtentorial disease and after the suboccipital operation. As a matter of fact after the suboccipital operation the postoperative improvement is either very marked, or so slightly marked as to make the separation of a group of "slightly relieved" cases more or less artificial.

Other things being comparably equal the good effect of a suboccipital operation, if the latter do exert a decompressive effect, is apt to be much more pronounced than that of a subtemporal operation. The difference is probably due to the fact that with subtentorial neoplastic disease the relative smallness of the posterior fossa, as compared with the cerebral, compels an earlier recognition of the disease and the performance of the operation at a time when the recuperation is more quickly and outspokenly possible and when mechanical disabilities are more easily correctable; the reverse is true with the supratentorial cases.

Effect of Decompression on the General Effects and Symptoms of Brain Tumor. Intracranial compression in brain tumor is, generally speaking, a slowly developing phenomenon. Far-reaching effects may, therefore, develop and continue for comparatively long periods of time; in this regard the phenomenon is vastly different from the quickly developing compression following cranio-cerebral injury. The exceptions to this rule are, however, not very uncommon. Then signs of compression appear quickly chiefly because of hemorrhage into the brain or tumor substance or into the ventricle; the symptoms include rapidly progressing stupor, increase in the papillo-edema, slowness of the pulse, etc. The ordinary slow form of compression is variously influenced as indicated subsequently in this communication. With acute forms of compression complicating tumor of the brain, especially in the variety in which hemorrhage occurs into the ventricle, the ordinary forms of decompression exhibit no benefit; the usual course of affairs includes a continual progression of the picture and exitus follows fairly rapidly.

The condition of the brain substance as exposed in the operative field has important bearings on the appearance of any subsequent good effect of the decompression in cases of neoplastic disease. There may be one of two conditions:

1. In one variety the brain is under a variable degree of tension under the unopened dura. In the most extreme of these the

degree of tension is such that there is very little, if any, give to the exploring finger as it presses on the dura; there is no pulsation. The slightest nick in the dura causes an instantaneous extrusion of brain tissue; a larger incision is followed equally rapidly by the progressive prolapse of the exposed brain substance, so that very quickly it forms a large projection above the surface of the dura. In the severe cases the cortex is anemic. In the moderate cases the surface of the brain has a deeper tint than normal, has a glazed appearance, is covered with dilated and distended pial vessels and feels firmer to the finger than it normally should. The tissue is extremely fragile. Bleeding occurs very readily at the slightest touch, or occurs spontaneously. At any manipulation, or in the extreme cases without any, the surface of the cortex fissures and ruptures and, very quickly, the brain substance becomes dark red and disorganizes completely, so that the characteristics of normal brain tissues are lost. These phenomena are more frequently seen in the subtemporal cases. In the latter there is consequently much difficulty in properly closing the wound. These changes are due to interference with the circulation in the cortex.

2. In other cases there are none or, at most, very little of these changes owing to the fact that there is no increase in intracranial pressure. In fact the gross appearances of the brain are those expected in normal individuals. In a very few I have thought that there was less than the normal present both as regards the relative size of the brain to its containing bony case and as regards the tension under which it functionates.

One would expect that the first of these two groups would contain the cases in which the maximum amount of good would follow the decompression. As a matter of fact, however, this is not so. It is very remarkable that the second group—those with very little, or no increase in intracranial pressure and with very little macroscopical change in the operative field contains the cases in which beneficial effects are more frequently seen. This is especially apt to occur with the subtemporal decompression cases.

The explanation of this phenomenon does not seem quite clear. In those with marked increase of intracranial pressure a possible explanation exists in the following. The increased tension and marked bulging of the brain is caused by an increase in the total cubic content of the brain (1) either because of simple swelling (Reichard), or hyperplasia (Spiller) accompanying an infiltrating type of tumor; or (2) because of the internal hydrocephalus; or (3) by varying proportions of both of these causes acting together. The increased intracranial pressure is due to an expansive force acting from the interior of the brain; and especially in the cases of internal hydrocephalus any relief which occurs is more than counteracted by an increased accumulation of intraventricular fluid. When the possibility exists of recognizing this state of affairs

before operation, the futility of doing subtemporal decompression is now recognized. In these cases of internal hydrocephalus good results can sometimes be obtained by doing a corpus callosum puncture.

I hazard the following explanation for the other cases—those with no bulging of the brain and with apparently no evident macroscopical change in the operative field. Possibly, in these cases the increased intracranial tension is due to some agency working externally to the brain—between it and the cranium. Possibly this agency is an increase in the amount of cerebrospinal fluid; not much would be needed to disturb the intracranial static conditions as brain tissue is physically incompressible. Such a mechanism would explain adequately, also, the appearance, referred to previously, in which the brain has given the impression of being slightly smaller than it ordinarily should be. The removal of a segment of bone and the opening up of the dura could, under such conditions, exert a very appreciable effect. I give the notes of one case in which, it seems to me, this viewpoint is corroborated by the postoperative facts.

In a patient with well marked general symptoms of brain tumor including a moderate grade of optic neuritis and extreme headache, the typical subtemporal decompression was done. There was no macroscopical evidence in the operative field of any increase of intracranial pressure and no bulging of the brain. However, an immediate marked beneficial effect followed including the complete disappearance of the headache. No appreciable bulging of the soft parts over the site of the decompression followed for some time. Then the area gradually began to bulge and the swelling grew larger and very tense and coincidently with this development the headache returned and grew progressively more marked. The bulging was due to an accumulation of cerebrospinal fluid and when the latter was removed by aspiration, the headache promptly disappeared, only to reappear once more as the fluid reaccumulated. This was repeated several times. The symptoms were unquestionably due to brain compression produced by a force acting from without.

The effect of intracranial compression has a direct and proportional relation to the competence or poverty of the general condition of the patient as determined by the condition of the circulation and the general state of the vital functions; these are not impaired appreciably in cases of brain tumor except when the compression is marked and has existed for some time; these usually indicate secondary effects on the medullary centers. After decompression any rehabilitation of these centers and any improvement in their functions are in direct proportion to the degree of decompressive effect.

*A. Effect of Decompression on Stupor and Drowsiness.* Four of the patients in this series were deeply unconscious, stuporous or comatose prior to the decompressive operation. Two of these were unlocalized cases of brain tumor; 1 had a tumor in the frontal region; and the fourth had a tumor of the thalamus. One of these (thalamic case) improved remarkably; 2 others were not benefited at all and died very shortly after operation; the fourth was removed from the hospital against our advice in a comatose condition, the operation not having been followed by any improvement, and, presumably died later at home. The first 3 were subtemporal, the last was a suboccipital operation. In our cases the stupor or coma had appeared fairly suddenly and had precipitated the operation. The experience seems to show that as a general rule a decompression will not influence a suddenly appearing stupor and that the latter can be classed in the great majority as an antemortem or terminal phenomenon of tumor of the brain. In an isolated case, however, a good effect can be obtained. The notes of one case may be of interest:

Hospital No. 154,635. A woman, aged fifty-five years, had the general symptoms of brain tumor. The symptoms included: (1) Marked headache; (2) marked rigidity of the neck; (3) marked Kernig; (4) weakness of the right facial nerve of the thalamic type; (5) weak knee and ankle jerks. Lumbar puncture demonstrated increased tension to the cerebrospinal fluid. The Wassermann reaction was negative. There was a papillo-edema of one diopter. The patient was unconscious though not as deeply stuporous as in the fatal cases referred to previously. Following a right subtemporal decompression there was marked improvement and the patient was able to be up and about the wards. The facial weakness lessened; the rigidity of the neck and the Kernig disappeared

Six other patients of this series exhibited various states of drowsiness; 1 had a tumor of the pineal gland and 1 of the frontal region; the others were cases of unlocalized tumor. The pineal patient showed no improvement; the frontal patient showed a slight grade of improvement so that questions could be answered even though with difficulty. In the others the drowsiness deepened to stupor and in 2 of these, explorations were done at a later period. In general the experience is not encouraging in regard to the effect of decompression on this symptom. The fact that there was a tendency in these cases for the drowsiness to deepen to stupor probably indicates that, as a general rule, the symptom is one which is not relievable by decompression and is a forerunner of deeper states of coma or stupor appearing as terminal phenomena of tumor of the brain.

There was 1 case of hypophysial tumor in the series in which drowsiness appeared as a new symptom after a subtemporal decom-

pression. There were other evidences of endocrine disturbance in this patient, including an amenorrhea; and I think that the drowsiness was probably related to the endocrine malfunction and was not an effect of the decompression.

*B. Headache.* In 43 per cent of the cases of subtemporal decompression, in which the operation was successfully carried out, the headache, which had been present before operation was not relieved; in 37 per cent it was partially relieved and in 18 per cent it was wholly relieved. In 20 per cent of the suboccipital cases, in which the operation was successfully carried out, the preëxisting headache was not relieved; in 40 per cent it was partially relieved and in 40 per cent it was wholly relieved. Headache is the one symptom which the patients complain of mostly and any improvement is very welcome. Fortunately headache is relieved, either partially or wholly, in a large proportion of the cases of brain tumor that are decompressed. Our statistics show that the symptom is more often relieved in cases of subtentorial, than in cases of supratentorial disease; and it corroborates the statement, made in the earlier part of this communication that the suboccipital cases are more apt to have the subjective symptoms relieved than the subtemporal cases.

*C. Effect of Decompression on Mentality.*[1] In the cases of brain tumor in which the clinical picture led us to suspect that the lesion lay in the frontal areas, decompressive operations had no effect upon the character of the disturbances of mentality and personality.

2. Effect of Decompression on Focal Symptoms. *A. Effect of Decompression on Spasticity.* Spasticity of some portion of the body was a prominent symptom in 6 of the patients. In 1 the right side of the body was held rigid; in a second a similar phenomenon was present at times and was associated with a temporary loss of speech. The first of these patients died after operation; the second was not improved. In the 4 other patients there was marked rigidity of the neck. Two of them had unlocalized tumors; both died after operation. In another, the tumor was in the posterior fossa and the spasticity was not relieved by the decompression. The fourth was a patient with a frontal lobe tumor; the rigidity of the neck promptly disappeared after operation and the patient's general condition was markedly improved. It appears that as a general rule spasticity is a rather unfavorable symptom and is not usually relievable by decompression. Except for the fact that the decompression resulted in a general relief of intracranial pressure, I have no explanation to offer for the good effect noted in the one case as opposed to the uniform absence of effect seen in the other cases.

[1] Changes in mentality are classified in this communication as general symptoms. It is to be remembered, however, that the symptom is frequently utilized as a contributory factor in the localization of a tumor in the frontal regions.

*B. Effect of Decompression on Convulsions.* One of the patients in this group was subject to epileptic seizures for twelve years prior to the onset of the symptoms of brain tumor. Another had generalized epileptiform convulsions only during the period in which the symptoms of brain tumor were present. A third patient had convulsive seizures limited to the right side of the body. A fourth patient had attacks of rigidity limited to the right side associated with loss of speech; later he developed convulsions.

In 2 of these patients fatalities occurred, the reason for which probably lay in the fact that an extensive exploration had preceded the decompression. In the others there was no improvement in this symptom. In this series of tumors of the brain, this symptom was not relieved by decompression; this agrees with the usually held view in regard to the effect of any form of operation upon well established epileptiform conditions.

*C. Effect of Decompression upon Muscular Weakness and Paralysis.* There were no cases of monoplegia or diplegia in this series of brain tumors.

There were 10 patients in whom a more or less pronounced weakness or paralysis of one side of the body was one of the symptoms of the clinical picture. Three of them died after the operation, 4 were entirely unrelieved and the remaining 3 were markedly benefited by the decompression. One of the last had had, originally, a spasticity and, in him, a change to a hemiplegia occurred as a sudden phenomenon; the promptness with which the decompression was done in this patient probably had much to do with the relief of the symptom. In our experience, when the patient is in good general condition and the weakness or paralysis has not been of too long duration, beneficial effects may be expected to follow a decompression.

*D. Effect of Decompression on the Reflexes.* In patients with brain tumors the condition of the reflexes follows no essential rule. Frequently, indeed, they correspond in the orthodox manner to the character of the lesion; but, just as often, the reflexes show an alteration of activity, which may or may nor be equal or unequal on the corresponding sides of the body and for which change no explanation is entirely satisfactory. In a similar manner abnormal reflexes are present or absent. After decompressive operations the experience goes to show that a similar absence of uniformity makes it difficult to draw deductions that would be of value in clinical neurological surgery. The condition of the reflexes present after operation, as differentiated from the condition present before operation, may show wide variations in both directions; and changed conditions of normal reflexes, or abnormal reflexes appear as new phenomena when they were not present prior to operation. Interpretations are made very difficult because the changed pressure conditions within the cranium resulting from the decompression are, in themselves, capable of producing these variations of the

reflexes aside from the presence or progression of the neoplastic lesion.

*E. Effect of Decompression upon Sensory Changes.* Sensory changes of one kind or another were present in a fair proportion of the cases of brain tumor; the changes varied from well defined areas of changed or absent sensation (fifth nerve distribution) to hemianesthesias. There were 5 patients in this series in whom these sensory abnormalities were present. One of these died following the decompression, 3 others showed improvement—in 1 of them the improvement was marked. The fifth was not improved. Taken by itself this symptom has little importance, but as a contributory factor in the entire clinical picture this class of symptom helps in the determination of the good, bad or want of effect of the decompression.

*F. Effect of Decompression upon Aphasia.* Eight patients were decompressed in whom some form of sensory or motor aphasia was prominent in the symptom-complex of brain tumor. Five of them died following the decompression; in all but 1 right-sided subtemporal operations were performed; in the exception, a suboccipital operation. In 1 of the patients the brain bulged considerably after the dura was incised. In all of the others, there was no marked abnormality to be seen in the operative field. Two of the patients who survived the operation were not relieved in any way; 1 was only moderately improved. In the patients of this series aphasia was generally associated with far advanced neoplastic disease of the brain; this explains the high mortality. Our experience indicates that the various forms of aphasia, both sensory and motor, are relatively little influenced by decompressive operations.

*G. Effect of Decompression upon Cranial Nerve Involvement.* Various grades of involvement of the different cranial nerves are common in the symptom-complex of brain tumor. Very slight changes in some of them are, frequently, evidence of generally changed intracranial conditions, whether these be due to tumor or to other lesions. Such slight changes are, apparently, not influenced by decompressive operations. When the cranial nerves are more markedly involved, they, of course are indicative of focal lesions, and help in the localization of the tumor. As will be shown in what follows; these are variously influenced by decompressive operations.

(*a*) *Effect of Decompression on Vision, Papillo-edema and Optic Nerve Atrophy.*[1] Eye symptoms are common in cases of tumor of the brain. In 18 of the patients of this series there were various grades of loss of vision in one or both eyes up to complete blindness and all of the patients had various grades of papillo-edema. Three of the patients died after operation; 6 were unimproved;

[1] For purposes of convenience, all eye symptoms are grouped together in this communication, although some of them are of a general nature.

3 were slightly improved; 1 was moderately improved. In 1 patient there was depreciated vision in only one eye which was greatly improved by the decompression.

In 2 patients there was an hemianopsia. One had, in addition, diplopia; and as a subtemporal operation did not benefit him and because posterior fossa symptoms developed, a suboccipital operation was performed with fatality. The second patient with hemianopsia was greatly improved by the subtemporal decompression. One patient, who was blind in one eye, was not relieved by the decompression. These cases show that the vision of many of the patients was markedly benefited by decompressive operations; those, who were, were usually the ones who had had failing vision for comparatively short periods; in those who were not improved, failing vision was present for a long time and the accompanying neuritic atrophy was unrelievable.

In brain tumors loss of vision is due either to direct pressure upon the visual pathways or to optic neuritis. Papillo-edema was present in 22 patients of this series, and varied from a slight blurring to a swelling of 6 diopters or more. In some, the papillo-edema appeared quickly under observation in the hospital and rapidly advanced to an extreme degree; in another group, the patients came into the hospital with a high grade of choked disc, which either did not vary appreciably from day to day, or showed tendencies to increase. In the last group the assumption was that the papillo-edema had been present for a considerable period of time.

Of the 22 patients, 8 died after the decompression. In 7 patients, in most of whom the symptoms were of long duration, the swelling of the discs did not diminish after the decompression during the period they were under observation (from three weeks to three or four months). Only occasionally will a long-standing papillo-edema lessen considerably after operation. In 5 of the patients, usually the more recent cases, there was considerable improvement. In 1 patient the swelling grew more marked after the decompression. As a general rule in those who are benefited after the decompression, the swelling recedes slowly and very soon reaches a stationary stage from which it does not vary appreciably. In a few, improvement is more rapid and the swelling disappears completely.

The facts regarding papillo-edema are shown more in detail in the appended table:

| Preoperative condition. | | Postoperative condition. | | | |
|---|---|---|---|---|---|
| Degree of nerve swelling. | Total number. | No improvement. | Improvement. | Increase. | Died. |
| Blurring | 1 | .. | .. | .. | 1 |
| 1 diopter | | | | | |
| 2 diopter | 5 | 3 | 1 | .. | 1 |
| 3 diopter | 4 | .. | 1 | .. | 3 |
| 4 diopter | 2 | .. | 1 | 1 | |
| 5 diopter | 5 | 3 | .. | .. | 2 |
| 6 diopter | 1 | 1 | | | |
| "Plus" | 2 | 1 | 1 | | |
| "2 plus" | 2 | 1 | 1 | | |

The amount of secondary atrophy of the discs, that already exists, is as is well known never lessened by the decompressive operation. This holds true even for those cases in which the changes are apparently at their very beginning when the decompression is done. When diminution of vision is due to advanced optic atrophy the disability may become more marked and may end in blindness even after a satisfactorily completed decompression. Because of the varying degrees of optic atrophy that are present, the improvement, or lack of improvement in vision which follows a decompression does not always correspond with the changes in the swelling of the discs.

(*b*) *Effect of Decompression upon Oculomotor Disturbances.* There was only 1 patient in this series with ptosis. There was no change after decompression either for the better or worse.

There were 6 patients with disturbances in the fourth and sixth nerves leading to various degrees of strabismus with diplopia. Two of the patients died; they were both cases of unlocalized tumor. A subtemporal operation had been done in 1; in the other a subtemporal decompression was later followed by a suboccipital operation. Two other patients with unlocalized tumors, were very much benefited by subtemporal operations. In the fifth patient the symptoms pointed to an involvement of the left thalamus and a subtemporal operation was followed by marked improvement. The sixth patient had subtentorial symptoms and was much improved by the operation.

Nystagmus was noted in 11 of the patients of this series; 5 of them were cases of cerebellar tumor, 1 of mid-brain tumor, and in the others, the tumors were unlocalized. Two of the patients with cerebellar tumor died. Nystagmus sometimes appears in the picture as an item of indefinite value and seems to be related to the general pressure disturbances within the cranium. In subtentorial disease, however, it usually has diagnostic significance. The influence of decompression upon the nystagmus was the following: In 3 cases there was no change, in 3 others there were various grades of improvement, in 1 the character of the nystagmus changed after operation, and in still another, a fine nystagmus appeared where none had been present before operation. Even in the cerebellar cases, in which this symptom is most apt to be present, there does not seem to be any definite relationship between the nystagmus and any improvement or lack of improvement which follows the decompression. Taken alone this symptom cannot be taken as a measure of the relief afforded by the operation; taken in combination with other elicitable data, it furnishes contributory evidence of the success or lack of success of the decompression. There is no absolutely reliable interpretation with which one can regard the symptom when it reappears as a new factor after decompressive operations.

(*c*) *Effect of Decompression upon Eighth Nerve Symptoms.* Deafness was present in 2 cases of cerebello-pontine angle tumor without any vestibular changes. Unfortunately, both of these patients died and no deduction can be made regarding the effect of decompression. There was 1 patient with vestibular changes in whom the condition remained unchanged after operation.

A very remarkable case was the following:

In a young boy with the symptoms of subtentorial brain tumor a suboccipital operation demonstrated a cyst of the cerebellum. The operation was practically a decompression. There was relief from the symptoms for more than a year after which the patient returned to the hospital with his old symptoms and the functional tests pointed to changes in the vestibular apparatus. The cyst was then aspirated through the area of bone defect with an immediate disappearance of his symptoms and a change to the normal in the vestibular apparatus as demonstrated by a repetition of the functional tests.

The observation is of value in indicating how, in specially favorable cases, one can prolong the good effects of a decompression. In cases of cyst or of internal hydrocephalus the reaccumulation of fluid causes a return of the symptoms of compression. The withdrawal of the fluid results in a corresponding relief of the symptoms.

In the cases of brain tumor in this series in which major focal symptoms were a part of the clinical picture, our experience shows that little hope of benefit may be held out to result from decompressive operations. Usually the disease is very far advanced, or its effects of so profound a nature as to lead to a fatality in many and to no benefit to the largest proportion of those who do not succumb.

Decompression for Cranio-cerebral Injury. The value of the various forms of operative treatment in cranio-cerebral injury were summarized in a previous communication.* From the point of view of the neurologist, as well as of the general surgeon, the opinion seems sound that conservative and expectant methods of treatment, whenever they can be safely employed, yield the best general results. We are unalterably opposed to operating indiscriminately upon all patients with cranio-cerebral injury. As the cases present themselves for treatment they seem to fall into one of three groups: (1) A large group the members of which always recover spontaneously; (2) a smaller group the individuals of which are almost certain to die with or without operative treatment and in which any form of treatment is futile; (3) a very small group in which the individuals present such border-line evidence as to make a policy of watchful expectancy advisable, but for whom one

* Wilensky, Am. Surg., October, 1915.

should be constantly on guard and in readiness to operate at a moment's notice. The final outcome in the individuals in the last group is always doubtful.

Irritative or paralytic focal symptoms which point to pressure upon definite cortical areas are important indications for operative interference. Isolated or irregular disturbances of neurological function existing alone in the absence of evidence pointing to involvement of definite brain areas do not constitute an indication for operation. They undoubtedly indicate some brain lesion the effects of which are of only a temporary nature and are recoverable from, or can be compensated by newly assumed functions in other regions of the brain. When the need for operation exists because of definite focal symptoms, the indication is to do an exploratory procedure over the suspected area and to rectify the pathology, if possible. When, as is most commonly so, the latter is a blood clot or depressed bone, beneficial effects are customarily seen and the symptoms promptly disappear; otherwise the operations are futile. In either case after left-sided explorations, contralateral subtemporal decompressions might conceivably be indicated to obviate the effects of any brain edema existing previously because of the original trauma, or occurring subsequently to operation as a consequence of the latter itself.

Focal symptoms, the causes for which are not demonstrable or removable during an exploratory craniotomy are not influenced in the slightest degree by decompression alone. They are usually due to destructive lesions of brain tissue and divisions of nerve trunks. This was well illustrated in one patient in whom the injury resulted in total blindness undoubtedly due to division of the optic nerves; a subtemporal decompression had absolutely no effect and the patient remained blind.

Operation is imperative in every case of cranio-cerebral injury which shows the signs of an advancing intracranial compression. Operation should be done in the early stages before there is evidence of medullary involvement. The nature of the procedure is determined by the suspected character of the injury and of its effects. Exploration is needed when hemorrhage is suspected in order to stop the bleeding and remove the accumulated blood, this being the commonest cause for a rapidly advancing compression. When the compression is due to massive brain edema the indication would be met by a decompressive operation. Frequently the differentiation is not possible, nor the side of the head which harbors the major lesion; in these a button of bone should be lifted out on the left side and if no accumulation of blood is found either outside or underneath the dura, a right-sided subtemporal decompression should be done. The prognosis is good in the milder cases, but small in lesions which cause extreme brain edema and compression; the latter are usually fatal.

In the cases of cranio-cerebral injury in this series, in whom decompression was done, the operations were performed in all but one of the cases for extremely urgent indications (compression) within a few hours of the injury. In an isolated case, the patient continued in stupor for five days without any apparent advance in the signs of compression; then, because there was a marked fall in the blood-pressure, the decompression was done with a fatality within a few hours.

In a previous communication[1] the value of lumbar puncture as a decompressive measure in cranio-cerebral injury was summarized. Experience showed that when the intracranial compression was of a high grade, lumbar puncture had no decompressive effect and other more active measures were sometimes necessary. In mild cases of compression it has been difficult for us to say definitely that the release of fluid from the subarachnoid space by lumbar puncture had helped in the amelioration of the symptoms, because in some mild cases in which the procedure was not carried out, spontaneous improvement followed. The good results which Alhaique, LeRiche and Albert obtained with lumbar puncture and probably explainable on this basis. The experience of Cushing and Gray is more in accordance with our own.

Decompression in Post-traumatic Epilepsy. There were 3 cases of epilepsy following cranio-cerebral injury, in whom decompressive operations were done. In 1, sufficient improvement followed the unilateral operations to warrant its repetition on the opposite side; the final result, however, was unsatisfactory. In another case with a depression near the median line at the vertex of the skull, the excision of the scar left a large opening which undoubtedly functionated as a decompression; in this case also no improvement followed. In another patient a subdural cyst was encountered which was drained through the decompression opening. The final result is not known in this patient.

In 1 case of epilepsy in a microcephalic child of twenty months, a considerable excess of cerebrospinal fluid was demonstrated at the decompressive (subtemporal) operation. Unfortunately the patient died of a meningitis. Our experiences with epilepsy have been small because the operations, for this indication, were done in much earlier times. Decompression is not now done for this indication.

In the earlier experiences decompressive operations were done in a few cases of Little's disease, and, perhaps under error, in cases of degenerative and inflammatory disease of the brain. The experience, however, was very small. Decompressive operations are not done for such indications now.

[1] Wilensky: Ann. Surg., October, 1919.

**Conclusions.** The mortality of decompressive operations is entirely dependent upon the gravity of the indication, the length of time the neoplastic disease had preëxisted and upon the general condition of the patient.

Generally speaking 57 per cent of the patients of this series in whom the operations were succesfully carried out were relieved in greater or less degree.

Any beneficial effect of decompression upon the symptoms of brain tumor is not always proportionate to the degree of the pre-existing increase of intracranial pressure.

Many of the cases of brain tumor studied in this series were admitted to the hospital in the terminal stages of the disease in which both general and focal groups of symptoms were well marked. As a general rule, it may be said that under such disadvantageous conditions the results are not good, even though in exceptional instances unexpectedly good results are obtained, more especially in the general symptoms and to a less degree in the focal groups of symptoms.

In cranio-cerebral injury the indication for doing decompression is seldom present.

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## BACTERIOLOGY OF ACUTE RESPIRATORY DISEASE DURING AN INTEREPIDEMIC PERIOD, WITH A DISCUSSION OF CONDITIONS FAVORING STREPTOCOCCUS EPIDEMICS.*

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During the winter of 1921–1922, the acute respiratory disease occurring in Philadelphia presented nothing of the epidemic character observed for these diseases throughout the United States during the winter of 1918–1919, so that an analysis of the bacteriological findings during the two periods appeared to be of especial interest in a study of the conditions determining the epidemic and the sporadic manifestations of acute respiratory diseases.

The findings presented for the more recent period represent the results of cultures made in the Bacteriological Laboratory of the Philadelphia General Hospital of specimens sent to the laboratory from the wards of this hospital treating acute respiratory diseases. They serve to indicate the nature of the acute respiratory infections in this hospital from October, 1921, to May, 1922.

* Read before the Philadelphia Pathological Society, May, 1922.

DIRECT CULTURES OF SPUTUM. In 28 instances, direct cultures of sputum were made on beef-infusion blood-agar medium. These represent a miscellaneous group of patients, 11 of whom were diagnosed clinically as pneumonia, the remainder being divided among such diagnoses as: Common colds, acute bronchitis, influenza, bronchiectasis, lung abscess and gangrene of the lung.

The findings in this group were:

| | Number of instances. | Per cent. |
|---|---|---|
| Pneumococcus | 12 | 43.0 |
| Type II | 1 | |
| Type IIa | 2 | |
| Type III | 2 | |
| Group IV | 7 | |
| Hemolytic streptococcus | 12 | 43.0 |
| Bacillus influenzæ | 20 | 71.0 |
| Streptococcus viridans | 18 | |
| Micrococcus catarrhalis | 19 | |
| Bacillus mucosus capsulatus | 2 | |

N.B.—By direct examination of stained preparations, spirilla were found in 2 instances and Bacillus tuberculosis in no instance.

The findings for the pneumococcus reveal particularly the failings of the direct cultural method, since with proper methods, an incidence of pneumococcus of from 20 to 25 per cent above the 43 per cent found here, can be demonstrated among normal individuals. The usual mouth types of pneumococci are shown in all except one instance, that of a pneumococcus Type II, so that, in view of the findings recorded in Table I, the findings presented above are certainly open to question from the standpoint of the reliability of the direct cultural method for demonstrating the pathogenic pneumococcus of a particular patient.

The direct culture method is more accurate for demonstrating hemolytic streptococci and Bacillus influenzæ. A demonstrated incidence of 43 per cent for hemolytic streptococci and one of 71 per cent for Bacillus influenzæ are significant findings in that they indicate that these organisms were lurking about in a large proportion of the acute respiratory infections. From the following tables an effort will be made to determine whether their presence was of as great a significance during this period as it was during the pandemic of influenza in 1918.

PNEUMOCOCCUS TYPINGS IN PNEUMONIA. Pneumococcus typings by the mouse method, with the sputa of 83 patients with pneumonia, gave the results recorded in Table I.

Type I pneumococcus occurred in about one-fifth of all the pneumonias, or in about one-fourth of the pneumococcus pneumonias.

This indicates a tendency to a return to the findings in pneumonia prior to the influenza epidemic.[1] The mouth types of pneumococci appeared here in a rather large proportion of instances. It must be remembered, however, that in this group were included

patients of all ages—many aged and suffering from chronic cardiac and kidney diseases—patients, in whom even an outstanding acute pneumonia is, in a sense, to be regarded as a secondary pneumonia. The high incidence of pneumococcus Type II (atypical), pneumococcus Type III and the inagglutinable Group IV pneumococci among such pneumonias is not surprising.

TABLE I.—PNEUMOCOCCUS TYPINGS—PNEUMONIAS.

| | Number. | Total. | Per cent. |
|---|---|---|---|
| Pneumococcus Type I | 15 | | |
| Pneumococcus Type I and IV | 1 | | |
| Pneumococcus Type I and B. influenzæ | 1 | Type I.....17 | 20.5 |
| Pneumococcus Type II | 3 | | |
| Pneumococcus Type II and III | 1 | Type II..... 4 | 4.8 |
| Pneumococcus Type II, atypical | 3 | | |
| Pneumococcus Type II, atypical, and B. influenzæ | 1 | Type IIa.... 4 | 4.8 |
| Pneumococcus Type III | 9 | | |
| Pneumococcus Type III and IV | 1 | | |
| Pneumococcus Type III and B. influenzæ | 1 | Type III....12 | 14.5 |
| Pneumococcus Group IV | 31 | | |
| Pneumococcus Group IV and B. influenzæ | 2 | | |
| Pneumococcus Group IV, Hemolytic streptococcus and B. influenzæ | 1 | Group IV....36 | 43.4 |
| Hemolytic streptococcus | 5 | | |
| Hemolytic streptococcus and B. influenzæ | 2 | Hem. strep... 8 | 9.6 |
| B. influenzæ | 4 | | |
| B. influenzæ and Streptococcus viridans | 2 | B. influenzæ..14 | 17.0 |
| Total | 83 | | |

Hemolytic streptococci occurred alone in 5 instances; with pneumococcus and Bacillus influenzæ in 1 instance; and, with Bacillus influenzæ in 2 instances; in all, in 8 instances, or in 9.6 per cent of the specimens.

Bacillus influenzæ occurred alone in 4 instances, and in combinations with pneumococci and streptococci in 10 instances—in all, in 14 instances, or in 17 per cent of the specimens.

Bacteriology of Empyema Following Pneumonia. Of the 83 patients with pneumonia listed above, 4 developed empyema. Five other instances of empyema, in patients entering the hospital with this condition were included.

TABLE II.—BACTERIOLOGY OF EMPYEMA FOLLOWING PNEUMONIA.

| | |
|---|---|
| Pneumococcus Type I* | 1 |
| Pneumococcus Type I and Bacillus influenzæ | 1 |
| Pneumococcus Type II* | 1 |
| Pneumococcus Type III | 1 |
| Pneumococcus Group IV | 2 |
| Pneumococcus Group IV and hemolytic streptococci | 1 |
| Pneumococcus Group IV and Bacillus influenzæ* | 1 |
| Hemolytic streptococcus* | 1 |
| Total: | |
| Pneumococcus | 7 |
| Pneumococcus and hemolytic streptococcus | 1 |
| Hemolytic streptococcus | 1 |

* Cases developing among the 83 pneumonias of Table I.

The noteworthy feature of the empyema following pneumonia was the predominance of pneumococci as the etiological agents. The one instance in which a hemolytic streptococcus occurred alone was in the case of a child developing otitis media, bronchial pneumonia and empyema complicating pertussis.

**Discussion.** In this hospital service, during the period of observation, hemolytic streptococci and Bacillus influenzæ did not appear to play prominent roles in the pneumonia and in empyema following pneumonia. Neither organism appeared to be especially pathogenic for mice, since, in the pneumonia typings, these organisms did not "come through the mouse" in any great number of instances. This was in direct contrast to what was almost the rule for these organisms during the influenza epidemic. Again, while hemolytic streptococci were present in a considerable number of sputa on direct culture, the empyemas following pneumonia were for the most part due to pneumococci. This indicates a lack of power for invasion on the part of the hemolytic streptococci. The lowered pathogenicity for white mice and the lowered incidence of hemolytic streptococci in the empyemas following pneumonia, appear to indicate that both of these microörganisms had lost their powers to invade deeply a pneumonic process. During the 1918–1919 influenza epidemic, hemolytic streptococci had acquired a deadly power to invade. This was also true of Bacillus influenzæ, which invaded the lung deeply, at times reaching the pleura, but rarely the blood stream in demonstrable numbers.

The question arises as to why the hemolytic streptococci and the influenza bacilli of the past winter differed in their abilities to produce complications in pneumonia, from that of their progenitors back in 1918–1919. What applies to one of these organisms may be said to apply to the other as well, so that this question may be answered if one explains why a hemolytic streptococcus in the natural exudates, or tissues of a person dying of a generalized streptococcus infection, upon direct inoculation into animals proves very fatal, while the same organism, after being grown even once in artificial culture, proves much less virulent. After a number of subcultures on a artificial medium, virulence is greatly lessened, but it can be restored in large part, by rapid, preferably mediate, transfer from one susceptible animal to another through a series of inoculations. For enhancing virulence by such a laboratory experiment, it is necessary that *the dose be massive at first*, that it sets up *an acute inflammatory process* and that *material from this acute inflammatory process at its height* be inoculated into the animal next in the series of transfers. In man, these conditions are fulfilled naturally by spontaneous transfer of infection during the building and maintenance of epidemics of acute respiratory disease.

Prevalence of Hemolytic Streptococci as a Factor in Determining Virulence. In throat cultures of 64 men, upon their

assembly from outlying country districts in November, 1918,[2] hemolytic streptococci were not demonstrated in a single instance. By multiplying the methods of examination, so as to include direct sputum cultures and cultures of the peritoneal exudate of white mice inoculated intraperitoneally with sputum, among 50 of these men, 3 were shown to harbor small numbers of hemolytic streptococci.

Levy and Alexander[3] found among recruits arriving at Camp Taylor, that 14.8 per cent of them harbored hemolytic microorganisms (not definitely identified as streptococci). Contrasted with these findings, the same observers demonstrated an 83.2 per cent prevalence of hemolytic streptococci among healthy men in Camp Taylor. Irons and Marine[4] reported a 70 per cent incidence of hemolytic streptococci among healthy men at Camp Custer.

During the same periods, it was shown by a number of investigators[5] that among patients with measles, the incidence of hemolytic streptococci, as determined by throat cultures, was lowest upon admission to the hospital wards and increased with the duration of the period in the hospital, at times to high levels. Where rigid ward management was instituted to prevent this dissemination,[6] some degree of success was occasioned, and the streptococcus complications were reduced; while in wards where there was cultural evidence of the rapid contact spreading of hemolytic streptococci among patients with measles, serious complications due to these microörganisms were always encountered in the greatest numbers, appearing usually after an interval of a week or ten days from the time of onset of the contact dissemination.

The evidence indicates wide variations in the incidence of hemolytic streptococci and that the segregation of individuals, whether sick or well, is attended by a dissemination of this particular group of upper respiratory bacteria. Attending this dissemination, particularly among individuals already suffering with acute respiratory disease, there is the appearance of streptococcus complications, frequently in epidemic proportions. And yet, a comparison of the numbers of individuals acquiring hemolytic streptococci in their upper respiratory tracts under such conditions, with that of those among them who develop clinical manifestations of infection due to hemolytic streptococci,[7] has shown that the latter is made up of only a relatively small percentage of the former. Thus, even with widespread dissemination, hemolytic streptococci do not develop the property of inciting serious pathological processes generally among all acquiring the microörganisms.

Except under epidemic conditions, hemolytic streptococci have a rather low pathogenicity for man, so that they, of necessity, become opportunists—gaining access to foci already injured—the open wound, the postpartum uterus, the pneumonic lung, etc.,

where they gain the ascendency and very quickly extend to mask the whole process by the seriousness of the infection set up.

When acute respiratory disease exists in epidemic proportions, and where patients are crowded together for treatment, active transfer of respiratory bacteria from individual to individual occurs. The recently traumatized respiratory tracts provide ideal fields for the transplantation and rapid development of the opportunists among the respiratory bacteria. Conditions are analogous then to the laboratory experiment for increasing virulence by rapid, successive passage through susceptible animals. In epidemics the non-immunes provide the means for this rapid transfer of infection, each in turn emitting a more deadly virus than he received. The epidemic wanes only when the non-immunes become fewer and more widely scattered, thereby decreasing the frequency of transfer. The virus persists for a time in convalescents and in the immunes gradually losing in virulence. The immunes further serve to maintain the endemic character of the virus. Epidemological facts bear this out, in that, during the rise of the epidemic prevalence of a disease, the greatest mortality rate is recorded, while after the peak of the epidemic passes, the mortality rate declines.

Carriers of Hemolytic Streptococci. The foregoing observations lead to a more definite consideration of the classification of individuals harboring hemolytic streptococci as recognized by throat cultures. The term "carrier" will be applied to such. Three distinct types may be defined:

1. *The Chronic Carrier.* This type of carrier is represented by a certain small number of individuals who may or may not have had acute clinical symptoms attending the acquisition of the streptococcus, but who thereafter carry small numbers of hemolytic streptococci over extended periods of time. In such cases "ragged," hypertrophied tonsils may furnish the opportunity for the organisms to continue growing more or less passively. Mild chronic inflammations may be kept up in part by such infections. Chronic carriers are likely to be distributed more generously among individuals with diseased tonsils. It is this type of carrier which maintains the endemic streptococcus manifestations. It is quite probable that from time to time there may be transient manifestations of acute infection in the chronic carrier, amounting to anything from symptoms of a "rough" throat to those of acute tonsillitis. In such cases, this carrier may, for a time, become,

2. *The Acute Carrier.* This type of carrier is represented by the individual who has active acute symptoms caused by hemolytic streptococci. Such carriers harbor larger numbers of the organisms than the chronic carriers; are more active foci of dissemination; and, presumably, have organisms of higher virulence. Throat cultures from this type of cases show very frequently almost pure

cultures of hemolytic streptococci. The acute carrier may carry the organisms for a short period during convalescence, or may carry them for longer periods, thus adding to the number of chronic carriers. It is this type of carrier which directs attention to the epidemic spreading of hemolytic streptococci. The number of these carriers is relatively small as compared with the total number of carriers which may be present in certain groups of individuals. Among patients under treatment for measles, or for respiratory diseases, a greater number of acute carriers develop than among individuals otherwise normal. Evidence has been presented[7] which indicates that this type of carrier develops with the initial appearance of the particular strain of hemolytic streptococcus in the throat. The number of these carriers appearing in any particular group varies directly with the widespread prevalence of,

3. *The Contact Carrier.* This class of carrier is represented by a large number of individuals who acquire hemolytic streptococci in their throats under conditions favoring aëro-contact dissemination, and who do not develop symptoms referable to the presence of such organisms. These carriers vary widely in numbers, from none at all to 75 per cent or 80 per cent of a community. The number is directly dependent upon conditions favoring the spread of infection from individual to individual. Fluctuations in the number of these carriers cause the wide variations observed in the incidence of carriers at different places and in different seasons. From this class arise the acute carriers under epidemic conditions. Members of this class appear to act as stepping stones in the building-up of virulence of hemolytic streptococci in any particular epidemic, since acute carriers begin to develop somewhat later than the beginning of the upward trend of the contact carriers.[8]

Theoretical Considerations in Regard to Carriers of Hemolytic Streptococci. The argument presented will be based upon conclusions and hypotheses suggested in this study and in the study of hemolytic streptococci in the ward treatment of measles.[9] The terms "acute," "chronic" and "contact" carriers will be used to apply strictly in accord with the definition of each of these as presented in this paper. A brief *résumé* of the deductions from the data collected follows:

The great variations in the prevalence of hemolytic streptococci in different communities are due to the fluctuations in the number of the contact carriers. These in turn develop under conditions favoring epidemic building and aëro-contact dissemination of the organisms.

Contact carriers spread streptococci to normal individuals, most of whom in turn become contact carriers. Contact carriers harbor the organisms for a comparatively short time. The rapid transfer of hemolytic streptococci, by means of the contact carriers, presum-

ably enhances the virulence of the strain transferred. Contact carriers after several weeks do not themselves appear to be prone to develop into acute carriers.

Acute carriers appear for the most part among individuals suffering from acute respiratory disease. These acute carriers begin to appear after there has been considerable development of contact carriers. Acute carriers develop from a recently acquired strain of hemolytic streptococcus. The strains of streptococcus harbored by the acute carriers are, perhaps, more virulent than those harbored by the other forms of carriers.

Chronic carriers serve in maintaining the endemic manifestations of hemolytic streptococci, and as such are the primary foci of dissemination in epidemic building.

Any attempt to account for the varied circumstances attending the development of these different kinds of carriers must be regarded as theoretical, and as such it should be based upon lines of reasoning following what is at present known of the nature of the organism in question. Little is known definitely of why an organism at one time produces grave infection while at another time it produces only a passive infection in the same or in different individuals.

We endeavor to explain these differences, on the one hand, by variations in virulence of the organism and by differences in the manner and in the size of the inoculation; while, on the other hand, we assume different grades of susceptibility to the infection on the part of the individuals. The former may be said to determine when the organism attacks; the latter whom it attacks. These conditions pertain especially to hemolytic streptococcus infections of the respiratory tract and their extensions.

1. *Virulence of Hemolytic Streptococci.* No one can doubt but that the streptococcus implanted upon a pneumococcus pneumonia, producing within forty-eight hours a phlegmon of the lungs and pleura with widely scattered areas of suppuration, septicemia and death, differs in virulence from the streptococcus ofttimes found in the throat of a patient throughout the course of his pneumonia. Differences in the resistance of individuals cannot account entirely for these discrepancies in the grade of the infection. Furthermore, we know definitely from animal experiment that the virulence of hemolytic streptococci vary and that virulence may be raised by frequent passage through animals.

Do the hemolytic streptococci found in throats vary in virulence? Why? In answer to these questions we need only apply the analogy between frequent passage of a strain of hemolytic streptococcus through experimental animals and its frequent transfer from individual to individual by natural means under conditions where men are crowded together. One strain may pass from *a* to *b* to *c* to *d* and so on almost indefinitely. It may grow passively in the throats of *a*, *b* and *c* and build in virulence slowly; but, sooner or later, it will reach a throat where local conditions the better

favor its growth, or the throat of an individual who has a lowered general resistance. Here it grows rapidly, produces symptoms, builds in virulence quickly, and is ready to produce sudden grave symptoms in the next individual furnishing a particularly good field for growth. These latter individuals are the acute carriers. They appear to carry organisms developed to higher grades of virulence, and are sources of immediate danger in wards treating acute respiratory diseases. These cases appear to arise because of the development of a heightened virulence in a strain of hemolytic streptococcus by its frequent transfer. In the process of this development, many individuals, such as *a*, *b* and *c*, must play a part without themselves being actually the ones to suffer. The delayed appearance of serious hemolytic streptococcus complications in a ward where numerous contact carriers have been developing, is in keeping with this argument.

The cases *a*, *b* and *c* are the contact carriers. Their range within wide limits of incidence is to be expected under this conception of epidemic building. When contact carriers begin to appear in a ward, we have the first danger signal. Serious acute streptococcus infections invariably follow.

What happens in this passage from *a* to *b* to *c*, etc., if an acute carrier, with, for example, a mild grade of tonsillitis, develops, and the opportunity for transfer of the infection is not at hand? An interruption in the building of virulence occurs. Gradually, the forces of the body defense gain the upper hand and the infection, so far as symptoms are concerned, subsides. The organisms may persist on repeated cultures, or they may disappear. In the one case we have a chronic carrier; in the other, the clearing up of an acute carrier.

What is the status of virulence of the organism in case it persists and the patient becomes a chronic carrier? Two possibilities suggest themselves: (1) The organisms may persist with sufficient growth and the maintenance of sufficient virulence to cause chronic disturbances, perhaps with occasional acute exacerbations. (2) The streptococcus may persist in a more superficial growth. It then becomes an organism growing more or less outside of the body, gradually losing in virulence and tending toward saprophytic properties. As such, it may grow even in the tonsil crypts. Growing thus indifferently, it sooner or later assumes the properties it possessed in patient *a* before any interchange with other patients occurred. There is no reason to believe that as a saprophytic organism, not again causing appreciable disturbances, it in any way loses its ability for growth in other throats on transfer to them by natural means; or that it ever loses that potential virulence which may develop into active virulence on frequent transfer. In fact, there are reasons to argue from analogy that, as it tends toward saprophytic properties it becomes less highly specialized, and requires less delicate handling to obtain growth

on transfer. The development of contact carriers would be the natural result when individuals are exposed to these chronic carriers of hemolytic streptococci of low virulence.

2. *Susceptibility of the Individual.* So far as is known, immunity to hemolytic streptococci is, in its strictest sense, relative. Immunity for a streptococcus of a given virulence may be developed, but cannot be regarded as absolute protection against a streptococcus of a higher virulence. Reinfection is common. Acute carriers, which develop a relative immunity sufficient to combat successfully the infection at its height, do not necessarily maintain such high degree of relative immunity, even though the streptococcus persists, and they become chronic carriers. A decline in the relative immunity following the decline in virulence of the organism, as it persists and tends toward saprophytic properties, might be expected. Such lowered relative immunity, while always sufficient to protect against the strain carried, might not adequately protect against a branch of the same strain, which had gone abroad, and by frequent transfer again assumed high virulence. With any organism capable of a great range of shifting virulence specific immunity can only be relative.

Epidemic Building of Contact Carriers and Its Control. The data at hand is sufficient to warrant a brief discussion of the hemolytic streptococcus situation in the army camps in 1917 and 1918. We have learned to appreciate the hospital ward situation with regard to hemolytic streptococci, but have failed to appreciate another very important consideration—the camp situation. If we look back in review of the conditions in camps where hemolytic streptococci caused the most trouble, we find that the camps themselves were highly saturated with these organisms carried by men on duty in whom practically no symptoms occurred. From Camp Taylor, an 83 per cent[3] saturation was reported among men on duty; from Camp Custer, 70 per cent.[4] At Fort Sam Houston,[5] 35 per cent is reported among admissions for measles, which signifies a higher camp incidence. It is obvious that where such conditions existed in a camp, any measures taken within the base hospital of the camp would be, in large part, inadequate in controlling the streptococcus situation. The data cited for men from outlying country districts,[2] and that from Camp Taylor, for men arriving at camp,[3] shows that hemolytic streptococci were not prevalent among such men. The conditions where 70 to 80 per cent of men in a camp carried hemolytic streptococci must have developed in the camp, and it is not difficult to appreciate how such conditions may arise there. The one fact of fundamental importance to be appreciated in this connection is, that hemolytic streptococci may spread quite as readily among healthy men as they do among patients in a hospital. This dissemination has not been recognized because the large majority who acquired hemolytic streptococci showed no symptoms. In other words,

they were simply contact carriers. Those who developed symptoms (acute carriers) came into the hospitals perhaps for these symptoms alone, but more frequently for some other acute infectious disease, and the part which the hemolytic streptococcus played was appreciated only after it had produced serious complications. At this time, the tendency was to place the entire blame on the hospital without going back to the real place of origin.

Camp conditions favored the epidemic building of contact carriers of hemolytic streptococci for a number of reasons.

Foremost among them was the bringing together of large numbers of men, many of country origin, and the crowding of them into barracks where the conditions, if anything, were even more favorable for the spread of respiratory infections than were those of the hospital ward, because the same precautions were not taken to prevent it. This was, of course, necessary in the handling of large numbers of men, but one must face the fact and place the blame for camp epidemics of respiratory infection primarily on this circumstance. If the real dangers of this procedure are fully appreciated, there are many practical precautions that could be instituted which would tend to lessen the danger. The epidemic building of contact carriers of hemolytic streptococci in a camp should be guarded against just as assiduously as the building of an epidemic of measles, or of influenza, because of the very serious way in which the streptococcus later modifies the complications of these diseases.

Another very important factor in the rapid building of large numbers of contact carriers was the intermingling of recruits and men who had been in the camp for a time. The figures already cited for camp populations and for recruits showed vast differences in the incidence of hemolytic streptococci in the two groups. Recruits when placed in contact with men, of whom 4 out of 5 are carriers of hemolytic streptococci, would immediately be "fired" with the predominating hemolytic streptococci. Not only were hemolytic streptococci always available, but also measles and influenza have been at hand for the susceptible recruit. Multiple infection, of course, would place him at a decided disadvantage. Rapid increase in the number of contact carriers, as has been suggested, may favor the building in virulence of hemolytic streptococci and should be considered. What could have better favored the epidemic building of contact carriers within the camp than the "installment" method of bringing recruits into camp, as was done under the operation of the draft? Draft A was brought into camp, and after allowing time for the men in this group to exchange mouth bacteria, and for contact carriers to reach their height, draft B was brought in, thus adding fresh fuel to the flames and allowing the contact carriers to build to a higher plane. Other drafts followed at regular intervals before the effects of the last draft had subsided. In this manner, the most ideal conditions

were furnished for epidemic building in the camp. Recently, these conditions have been reproduced in epidemiological studies among experimental animals in the laboratory.[10] Records show that each of these drafts of recruits had its own quota of measles, of influenza and of pneumonia. From a knowledge of the rapid spreading of hemolytic streptococci by contact, it is only fair to assume that each had its own quota of contact carriers of hemolytic streptococci. It appears that a camp incidence of 70 to 80 per cent of contact carriers could have been built up only under such ideal conditions. The holding of the different drafts apart as entirely separate units until the throat bacteriology of the newest draft could have been interchanged and become more or less stabilized, would of itself have been a great factor in preventing such epidemic building.

A third consideration, which amounts to much the same, was the mingling of country and city recruits in the same organizations. Because of the congested conditions of city life, the throat bacteriology of urban recruits quite likely is more varied and complex than that of recruits from country districts, so that a mixing of the two groups would add impetus to the spread of mouth organisms among the recruits from the country.

**Summary.** The findings presented represent a study of acute respiratory diseases during an interepidemic period.

The pneumococcus is returning to its own in dominating the field in sporadic pneumonias.

Hemolytic streptococci and Bacillus influenzæ are present, existing as endemic viruses at their lower planes of virulence, yet ever ready to flare up as deadly factors in the complications of epidemic pneumonia, should it return.

Three types of carriers of hemolytic streptococci are defined.

In the building of epidemics of acute respiratory disease, many individuals who themselves escape the ravages of the disease, are concerned as elementary transfer agents in the preparation of a virus (*e. g.*, hemolytic streptococci) of sufficient virulence to attack with alarming surety.

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# A ROENTGENOLOGICAL STUDY OF TUBERCULOSIS OF LUNGS AND INTRATHORACIC GLANDS IN INFANCY AND EARLY CHILDHOOD.

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The high incidence of unsuspected tuberculosis in respiratory infections is strikingly brought home to the pediatrician conversant with an active roentgen-ray service. The routine application of roentgen-ray laboratory facilities serves to cut down the percentage of avoidable error in final diagnosis and helps materially in prognosis.

It is difficult to apply our knowledge of tuberculous changes gained through experience with adult pathology. In the infant early changes are much more massive, reactions more intense, and a definite predilection of the lymphatic structures to disease confuse exact interpretation of actual pathology. Acute serous changes predominate. Chronicity is the exception. The early stage may be of difficult differentiation and only the subsequent course enlightens us as to the true etiology. Our classification shall be a simple one as it strikes a roentgenological observer. Generally speaking, changes fall into two headings: (1) Those involving the glands; (2) those attacking the parenchyma.

1. *Glandular Changes.* These fall into three subdivisions, namely:

(*a*) Paratracheal.
(*b*) Perihilus.
(*c*) Peribronchial.

Finer classifications can be made, but a fusion of these processes is so general that for working purposes more divisions are cumbersome.

2. *Parenchymal Changes.* These are subdivided into those involving:

(*a*) Lungs.
(*b*) Pleura.

**Glandular Tuberculosis.** Generally speaking, we have found that the paratracheal glands are involved earliest in life, the perihilus somewhat later and the peribronchial latest in childhood. Sometimes the process confines itself to one region, occasionally more than one. In this case one site may be heavily involved and the other or others slightly so. Eventually an entire chain is affected.

*Early Changes.* Such a plate shows in the upper mediastinal region an evenly outlined shadow, rounded or elongated, depending

upon the degree of congestive reaction and the number of glands involved. Smooth contours and unilateral shadows of even density characterize this stage. If the process is perihilus or peribronchial, the changes apply to these respective regions.

Conflicting shadows in this region are neoplasms and congenital anomalies, which are very rare, and thymus, which in contradistinction is usually bilateral, and in good plates shows a fine leaf-like arborization.

Differentiation from non-tuberculous processes at this stage is difficult and only subsequent events can clear up the diagnosis.

The second stage of changes, and that of more characteristic pathology is that due to necrosis and fibrosis. Outlines are irregular and blotchy, and so-called mottling is present. Occasionally, one may notice a zone of parenchymal congestion, and when these various changes are simultaneously present, the diagnosis of tuberculosis is probable. The ultimate outcome of these changes has been twofold; in one type, a retraction of shadow takes place, with an increase in density and an accentuation in the mottling; in the other type, a definite progression to parenchymal involvement with a picture which falls under the heading of mixed parenchymal tuberculosis.

**Parenchymal Tuberculosis.**

1. Pleural.
2. Pulmonary.

*Pleural Changes.* These have been of two types; an intralobar effusion, secondary as a rule to hilus gland pathology. The process is sharply defined usually between the upper and middle lobes. It subsides, as a rule, leaving a definite transverse, linear scar. However, not all linear scars are tuberculous.

The second type of pleuritis is usually found as a complication of a diffuse, pulmonary involvement, the picture of which overshadows pleuritic changes. These changes are either serous or fibrous. Serous deposition is usually in small quantities. Fibrous depositions take place in the form of fine filament-like bands or in rare instances, a diffuse, thick, plastic mass, not unlike that seen in the adult pleuritis. All combinations of these two types usually are present. At no time have we seen a primary, uncomplicated tuberculous serous or fibrinous pleurisy.

*Pulmonary Changes.* There are three types: (1) Bronchitic; (2) bronchopneumonic or lobular; and (3) lobar.

1. The bronchitic type is but a stage of pulmonary changes observed early in a general miliary infection, when the tubercle's growth is barely discernible by plate methods and when death intervenes before full growth of tubercle has taken place. At this time, markings are accentuated and somewhat congested in appearance. This type of change is also seen with general miliary

abscesses, due to a terminal sepsis. Should the disease be prolonged, the process merges into the secondary bronchopneumonic type.

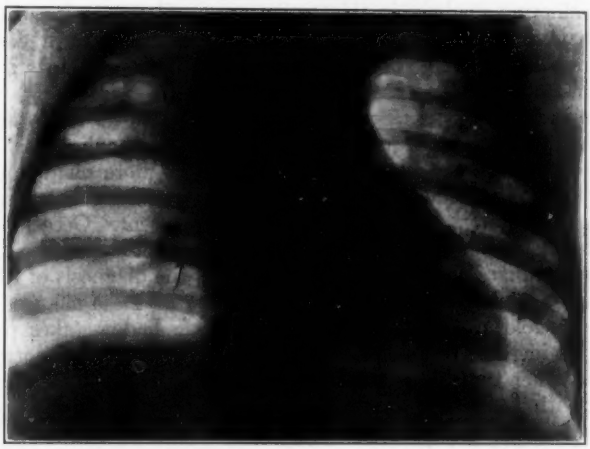

FIG. 1.—An example of annular shadow in an infant with a concomitant bronchopneumonic process. Arrow points to lower border of shadow.

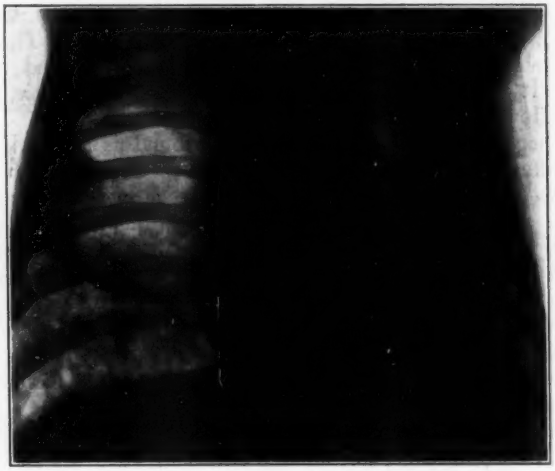

FIG. 2.—Diffuse terminal miliary tuberculosis grafted upon a chronic one. Terminal condition is an immense cavity with a diffuse involvement.

2. The bronchopneumonic or lobular type may be a bronchopneumonia from the very beginning, or it may be, as described

above, a stage in the bronchitic type. The lobular type may be an isolated area of involvement in any of the lobes, more often the

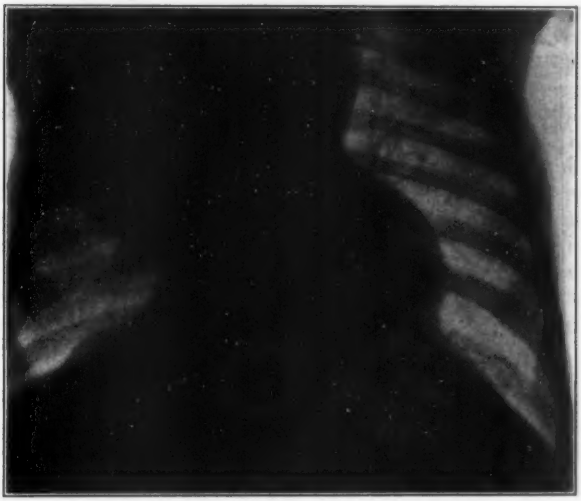

FIG. 3.—An example of paratracheal glands and a right sided perihilus pneumonia.

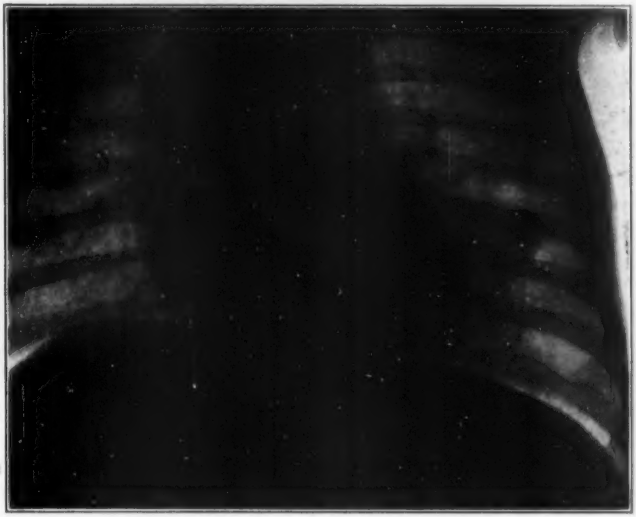

FIG. 4.—A perihilus, bronchopneumonic process of the left lower lobe; likewise a diffuse and unilateral enlargement on the right side, suggesting a universal involvement of all glands on this side.

lower, and usually perihilus in site, or there may be a general diffuse extensive involvement, no area larger than ½ cm. in diameter. In the first instance the parenchymal process may retrogress, the glandular condition partially subside and a period of latency vary-

FIG. 5.—An example of hilus tuberculosis with right-sided paratracheal glands.

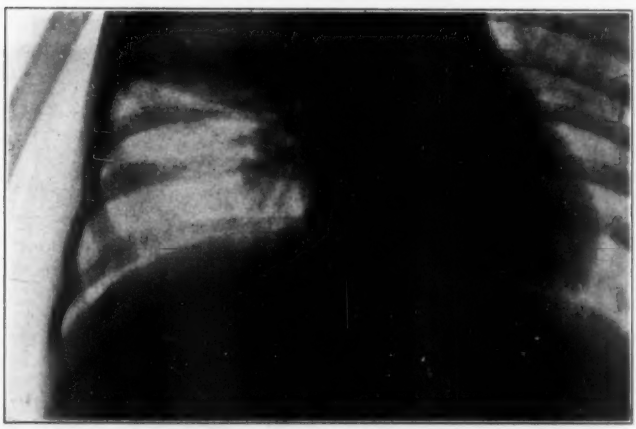

FIG. 6.—An example of a cavity which occupies the entire right upper lobe, the terminal result in the case of tuberculous pneumonia.

ing in time may intervene, followed by a flare-up of pathology. This may be repeated and go into a characteristic tuberculous process or subside completely without any parenchymal scars. The glands, however, always bear traces. This is the recurrent

perihilus, pneumonic type, the differentiation of which is so difficult and the exact diagnosis so often overlooked.

In the cases of diffuse involvement the whole lung is so riddled that few areas of normal tissue are distinguishable. Complicating this picture, are minute effusions, fine fibrous bands, and occasionally localized areas of thickened pleura. A plate of this type shows every conceivable pulmonary change; small cavities, glands, large areas of amalgamated pathology, and occasionally small areas of spontaneous pneumothorax due to ruptured pulmonary tissue.

3. The lobar type or massive tuberculosis is probably the rarest of all types. We have had instances of not only involvement of one entire lobe right from the beginning, but we have seen an entire lung disabled, so that it was impossible to eliminate the diagnosis of effusion, so similar was the plate pathology. Only repeated negative aspirations and autopsy findings overcame our hesitant interpretation. The course of this type of pneumonia was in two directions.

In one type, a retraction of pathology to a small area and a definition of the process to only part of the lung takes place. This is an instance of the massive response of the organism where the actual area of involvement was considerably less than the early changes depicted. The process now assumed a characteristic and definite evolution to a central area of softening and a typical abscess cavity. Practically no fibrous walling off takes place, and if the condition is permitted to continue its way, the abscess breaks through and we see a localized pneumothorax. Abscess cavities may be single or multilocular, minute or the size of the better part of an entire lobe. If, however, as in the second type, the infection is overwhelming and death intervenes early in the disease, the process may end with a lobar involvement without abscess formation. This shadow is almost liver-like in its quality.

In all types the other lung may remain comparatively free without any determinable changes for a considerable time after the original process has been seen, but inevitably involvement of one type or other takes place. The guiding post in diagnosis in the early stages is the usual concomitant glandular involvement. Prognosis, of course, is bad.

*Annular Shadow.* We mention this because we have had two instances of this present in infancy; in one case with a bronchopneumonia and glandular changes suggestive of tuberculosis, and in the other, without any tuberculous pathology. There are no tuberculous stigmata, and at the last observation, it is still present. Inasmuch as the significance of this is *subjudice*, we present this evidence for what it may be worth.

We take without question the use of the roentgen-ray as an adjuvant to exact diagnosis of tuberculosis in the later years of life. Why not broaden its application to infancy and the pre-

school age? Modern medicine, with its emphasis upon *prevention* should avail itself of this assistance. Surely, there are no technical difficulties, as any patient roentgenologist can prove. If the interpretation is still groping, then only wider experience can overcome that lack.

For prognosis its value is incalculable. For assistance in exact diagnosis in infancy when clinical signs are confusing, we cannot overestimate its value, not only in tuberculosis but in respiratory infections of all kinds.

**Conclusion.** Tuberculosis in infancy is of two types, acute and chronic; the first is the more common of the two and may be subdivided into:

1. Tuberculosis of the glands.
2. Tuberculosis of the lungs.

In both cases, by means of roentgen-ray follow-up, the process can be shown to have a definite and fairly orderly sequence, a period of congestion, a period of retraction or spreading, complete or incomplete and a period of softening.

While it is true, in the early or congestive stage, the process may not be distinguishable from any of the acute infections, in the second stage, of retraction or expansion, the condition assumes characteristic appearances and in the third stage, of pathology, there should be no difficulty in diagnosis.

For exact visualization of these changes, repeated and roentgenographic investigation is absolutely essential.

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## CARCINOMA OF THE ESOPHAGUS.

By Porter P. Vinson, M.D.

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From August 1, 1919, to August 1, 1921, 154 patients suffering from cancer of the esophagus were studied in the Mayo Clinic.* I have been able to trace all but 2 of these patients through the coöperation of their friends, relatives and attending physicians. A critical analysis of the group is herewith submitted.

As pointed out by Waggett, Janeway, Guttman, Held, Portis and others, malignant disease in the esophagus is much more common than is generally believed. Guttman and Held assert that from 5 to 7 per cent of all cancers found in man are in the esophagus, and Portis believes the percentage to be 20. In spite of this, reports of cases have been rather isolated, owing probably

* Sixty-two patients having malignant lesions at the cardia involving the lower esophagus secondarily, seen during the same period, have not been included in this series.

to unsatisfactory treatment and hopeless prognosis. In 1920, Turner reported a diagnostic-statistical study of 140 cases of the disease. The majority of reports have come from those interested in some particular form of treatment.

**Symptoms.** The symptoms depend largely on the stage of the disease. In the early stages there is usually slight dysphagia when swallowing solid food, and as the lesion progresses soft foods and finally liquids become obstructed. Food obstruction is progressive with no remissions. The onset of dysphagia is usually gradual, but may be sudden. At the time the first symptom manifests itself the disease is, of course, well advanced so there is very little hope of making an early diagnosis, although in many cases it can now be made much earlier than in the past.

Hiccough is an early and not infrequent symptom. Regurgitation is common, but is usually brought on voluntarily to rid the esophagus of obstructing material; it is rarely delayed, as in cardiospasm, and as there is usually very little dilatation of the esophagus above the stricture, the amount of food and mucus regurgitated is not large. Blood streaks in the regurgitated material are not commonly noted. Where the lesion is high in the esophagus, the constant expectoration of thick, tenacious mucus is very annoying. Because of food restriction there is continual loss of weight; the patients are rarely cachectic, yet there is a characteristic haunted facial expression. Pain may be present in the early stages, but is usually a late manifestation. It is located substernally, sometimes in the epigastrium, and may or may not accompany the act of swallowing. Occasionally hoarseness, due to cord paralysis from pressure on the recurrent laryngeal nerve from the primary growth or from metastatic cervical glands, may be the first manifestation. Hemorrhage and perforation are often terminal events. The average duration of symptoms in the series of 154 cases was seven months; the shortest duration was three weeks.

**Age of Patients.** One hundred twenty-seven of the patients were men and 27 women, a ratio of about 5 to 1. Almost all of the patients were more than forty years of age (Table I). Only 7 were less than forty, and the youngest was thirty-four years.

TABLE I—AGE INCIDENCE.

| Years. | Patients. |
|---|---|
| Youngest 34 | 2 |
| Oldest 77 | 2 |
| 31 to 40 | 7 |
| 41 to 50 | 31 |
| 51 to 60 | 61 |
| 61 to 70 | 38 |
| 71 to 80 | 17 |

**Location and Incidence of Lesions.** Syphilis of the esophagus rarely occurs, and it is doubtful if it was a factor in any of the cases

in this series. Eight of the patients had had syphilis, but only 4 had positive blood Wassermann reactions; the test was made in 87 cases.

Table II shows the occupation of the patient. Most of them gave histories of bolting food, meals at irregular intervals, and many used alcohol to excess. Dental sepsis was almost always present.

TABLE II—OCCUPATIONS.

| | Patients. | | Patients. |
|---|---|---|---|
| Farmer | 33 | Engineer | 1 |
| Home | 25 | Flagman | 1 |
| Laborer | 19 | Foreman | 1 |
| Merchant | 10 | Inspector | 1 |
| Clerk | 6 | Janitor | 1 |
| Not stated | 6 | Lawyer | 1 |
| Bookkeeper | 4 | Lumberman | 1 |
| Carpenter | 4 | Machinist | 1 |
| Miner | 4 | Minister | 1 |
| Bricklayer | 3 | Nightwatchman | 1 |
| Butcher | 3 | Orderly | 1 |
| Teamster | 3 | Papermaker | 1 |
| Baker | 2 | Peddler | 1 |
| Druggist | 2 | Physician | 1 |
| Shoemaker | 2 | Plumber | 1 |
| Architect | 1 | Salesman | 1 |
| Boilermaker | 1 | Stenographer | 1 |
| Boxmaker | 1 | Tailor | 1 |
| Builder | 1 | Teacher | 1 |
| Cabinetmaker | 1 | Waiter | 1 |
| Car repairman | 1 | Writer | 1 |
| Cook | 1 | | |
| Total | | | 154 |

The location of the lesion varied according to sex. Sixteen of the 27 women had lesions of the introitus, whereas 64 of the 127 men had lesions located from 27.5 to 37.5 cm. from the incisor teeth (Table III).

Twenty-one of the patients had metastasis; 11 were from lesions of the introitus. Nine of the patients had metastasis to the cervical glands, 1 to the lungs, and 1 to the substernal glands. Of the 10 other patients metastasis to the lungs was evident in 5, to the cervical glands in 4, and to the cervical glands and wall of the chest in 1. Seven of the patients were women and 14 were men. The apparent greater tendency for metastasis to occur in cases of lesion of the introitus is probably owing to the fact that the cervical glands were usually involved and the condition more easily recognized. As the majority of the women had lesions of the introitus, the relative increase in metastasis in this sex may be more apparent than real. Turner does not consider metastasis in his cases, but he shows very strikingly that the postcricoidal cancers predominate in women. Ninety-eight of the 140 lesions reported by him were located in this area and 85 of the 98 patients were women.

TABLE III—LOCATION OF LESION.

Distance from incisor teeth, cm.

| Cm. | Cases. | Women. | Men. |
|---|---|---|---|
| 18.75 | 27 | 14 | 13 |
| 20.0 | 8 | .. | 8 |
| 21.25 | 3 | 1 | 2 |
| 23.75 | 4 | .. | 4 |
| 25.0 | 3 | 1 | 2 |
| 26.25 | 4 | .. | 4 |
| 27.5 | 6 | .. | 6 |
| 28.75 | 4 | .. | 4 |
| 30.0 | 4 | 3 | 1 |
| 31.25 | 3 | .. | 3 |
| 32.5 | 17 | 4 | 13 |
| 33.75 | 7 | .. | 7 |
| 35.0 | 9 | .. | 9 |
| 36.25 | 10 | .. | 10 |
| 37.5 | 5 | .. | 5 |
| 38.75 | 4 | .. | 4 |
| 40.0 | 5 | .. | 5 |
| 41.25 | 2 | .. | 2 |
| 42.5 | 2 | .. | 2 |
| 43.75 | 1 | .. | 1 |
| 45.0 | 1 | .. | 1 |
| Upper third | 3 | 1 | 2 |
| Middle third | 6 | .. | 6 |
| Lower third | 15 | 3 | 12 |
| Not stated | 1 | .. | 1 |
| Total | 154 | 27 | 127 |

A history of previous trauma to the esophagus was not elicited in any case, but in a recent case there had been a burn of the esophagus from lye in infancy.

One of the patients had had cardiospasm with dysphagia for nine years. He had a widely dilated esophagus with obstruction of the barium meal at the cardia (Fig. 1). Esophagoscopic examination revealed epithelioma on the anterior wall of the middle third of the esophagus.

**Diagnosis.** The diagnosis of carcinoma of the esophagus is, in most cases, relatively easy. Plummer, however, has called attention to frequent errors in diagnosis, and Miller, Hyman and Janeway have recorded the fact that these patients may show postmortem lesions unsuspected during life. The history is of the greatest importance in recognizing malignant esophageal disease. The cautious passage of a blunt olive on a whalebone staff guided to the obstruction by a previously swallowed silk thread is also of value and is of the utmost importance when forcible dilatation with sounds are employed in the treatment (Fig. 2). When sounds are passed through the stricture, using the wire spiral as a guide on the thread, a pink tinged mucus, sometimes foul smelling, is usually noted on the spiral when it is withdrawn; this is almost pathognomonic of cancer. Roentgenograms were made in most cases in the series, and when the obstruction was irregular in out-

line, the diagnosis could almost be made on this evidence alone. However, the roentgen examination must not be accepted as infallible; in 4 of our cases a most careful examination failed to reveal obstruction in the esophagus. Esophagoscopic examination may clear up a doubtful diagnosis but neither is this infallible. Unless ulceration is evident it is probably not wise to remove a section for microscopical examination. Not infrequently the tissue removed reveals inflammatory changes only, when there is an underlying carcinoma.

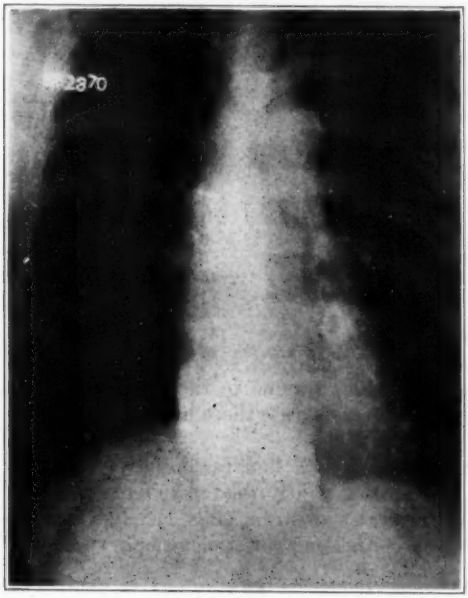

FIG. 1 (A332,370).—Cardiospasm complicated by epithelioma at the middle third of the esophagus.

Carcinoma of the esophagus must be differentiated from all other lesions producing dysphagia. However, about one-half of all esophageal lesions are due to carcinoma. Ninety per cent of patients more than forty years of age, without a history of previous trauma, who have had dysphagia for a year or less, have malignant obstruction in the esophagus. When these facts are considered, errors in diagnosis should be infrequent.

**Treatment.** The present mortality from malignant esophageal disease is practically 100 per cent, and therefore treatment should be directed along the lines of greatest palliation. Successful surgical removal of growths located at the introitus have been reported by

Turner and Wallace. Torek[35] has reported the successful removal of the growth in the middle third of the esophagus. Meyer, Bengolea and Lilienthal have reported resections in this location, but

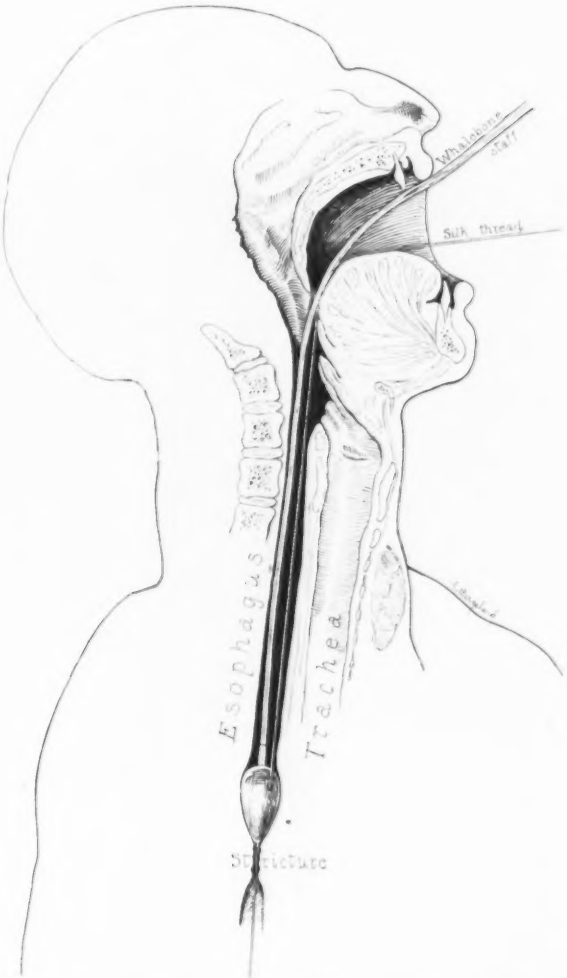


FIG. 2.—Method of measuring distance of obstruction from incisor teeth.

the benefits obtained were temporary only. Torek, in 1915, also reported resections of the lower esophagus and cardia by Völcker, Kümmell and Zaaijer. Hedblom recently reported a resection of the

lower esophagus and cardia for carcinoma. In 1917, W. J. Mayo performed a successful total gastrectomy removing 16 mm. of the esophagus.

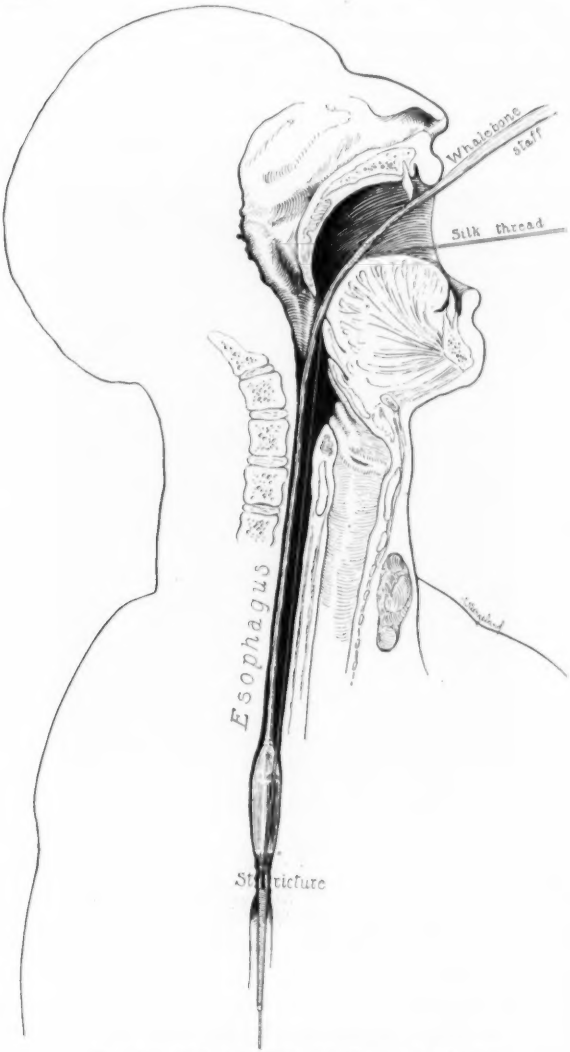


FIG. 3.—Method of dilating malignant stricture.

Surgery for the cure of these lesions is still in the experimental stage. Early diagnosis may bring about more cures, but the opera-

FIG. 4.—Esophago-tracheal fistula due to carcinoma. The thread is shown passing through the fistula into the trachea.

tion at best is formidable. Gastrostomy is advocated by many, particularly Jackson, Robson, Kreuscher and Lane, while Maclay decries this operation, particularly in the late stages of the disease. There are certainly just criticisms of the operation. The patients and relatives are usually very unhappy afterward, and if the disease is in an advanced stage the mortality is strikingly high. The operation really does not prolong life to any appreciable extent. Four of the patients had had this operation before coming to the clinic, and 11 had the operation at the clinic. Five of the 11 died within a week, from hemorrhage or infection; the average duration

FIG. 5 (A322,470).—Esophago-tracheal fistula due to spontaneous perforation of a malignant esophageal stricture. Outline of bronchial tree is shown by the ingested barium meal.

of life after operation in the cases in the series was three months. Esophagostomy has been suggested if the lesion is high in the esophagus, but this would seem less desirable than gastrostomy.

Radium and roentgen-ray treatments have been employed extensively, but no cures have been reported although palliation has evidently been obtained in many cases. Fifteen of the patients in the clinic were treated with radium and roentgen-ray, by the technic described in a previous paper. The most favorable cases were selected for this study, but the results obtained were altogether disappointing. One of the patients lived for twenty-three months

after treatment, but the average duration of life was only eight months. The patients received from 2000 to 3000 mg. hours of radium. Recently this dosage has been increased to 3600 mg. hours without definite benefit. Intubation of the malignant stricture has been done in some instances, but not in the cases seen at the Mayo Clinic.

Although no form of treatment will prolong life for many months, or give great relief from dysphagia, it is the experience in the Clinic that the dilatation of the carcinomatous stricture with olives, graduated sizes, gives the greatest palliation (Fig. 3). Those

FIG. 6 (A391,159).—Carcinoma of the esophagus with marked distortion of the lumen of the stricture. The blind passage of sounds in such a case will likely result in perforation.

objecting to this form of treatment say that it is dangerous, that it hastens metastasis, and that it does not relieve symptoms. In 125 patients in this series the esophagus was forcibly dilated and 3 deaths resulted. Many of the patients had several dilatations. No evidence has been obtained to suggest that dilatation in any way hastens metastasis. When an esophageal carcinoma is dilated to 45 F. immediate relief from dysphagia almost always follows, and this palliation lasts for from six to eight weeks. Further dilatations will usually afford relief, but of shorter duration. The average duration of life in this series after dilatation was five months. One patient not included in the series lived for three years

following a single stretching. He finally died from generalized metastasis.

All of the dilatations were done with sounds devised by Plummer and according to his technic. A word of warning should be given in reference to the swallowing of the silk thread used as a guide in passing sounds. In cases of esophago-tracheal fistulas the thread will pass into the trachea and will be coughed up and swallowed again. In this way as many as ten or more strands of the thread may become entangled as is shown in Fig. 4. If the thread is not removed it may produce edema of the larynx, necessitating tracheotomy. Removal of the thread is a very tedious process.

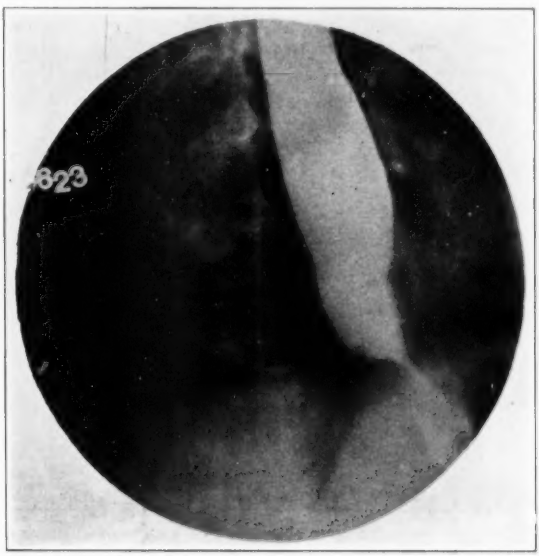


FIG. 7 (A386,623).—Carcinoma of the cardia with diffuse dilatation of the esophagus resembling cardiospasm.

Eleven patients of the series were not treated and the average duration of life was three months.

**Conclusions.** 1. The incidence of carcinoma of the esophagus is higher than is generally believed.

2. The symptoms are ordinarily quite characteristic and their duration from onset to death is one year.

3. Men are much more susceptible than women, the ratio being 5 to 1.

4. Carcinoma of the esophagus is rare in persons under forty years.

5. In men the lesions are most common in the middle third of the esophagus, whereas in women the growths predominate in the upper third.

6. Demonstrable metastasis is infrequent but occurs more often with lesions of the introitus and, therefore, is proportionately more common in women.

7. Previous trauma to the esophagus is not a definite factor in the causation of malignant esophageal disease.

8. The history of the case, and the cautious passage of sounds, are the most valuable factors in making a correct diagnosis.

9. The roentgenogram is of value in making a diagnosis, but is not infallible.

10. The esophagoscope is of limited value in diagnosis and treatment.

11. The mortality in untreated cases is 100 per cent, and surgery offers very little as a curative or palliative measure.

12. Radium or roentgen-ray therapy is practically valueless.

13. Forcible dilatation of the malignant stricture gives greater relief than any other form of treatment.

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# INTESTINAL INDIGESTION IN ECZEMA AND PSORIASIS.*

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(From the Skin Clinic and read before the Staff of the Massachusetts General Hospital.)

**I. Foreword.** "Man, who is the servant and interpreter of Nature, can act and understand no farther than he has, either in operation or in contemplation, observed of the method and order of Nature. The knowledge and power of man are coincident; for whilst ignorant of the cause he can produce no effects; nor is Nature to be conquered but by submission. And that which in speculation stands for the cause is what in practice stands for the rule." Bacon.[1]

**II. Intestinal Indigestion.** In this period of advanced civilization in which complex conceptions and ultra-scientific theories are sometimes elaborated, man is after all—what he has been and always will be—merely a servant and interpreter of Nature. He is the most highly perfected animal, yet like all lower forms is subject to the natural laws already made known and interpreted. These laws are just, but exacting and economical in operation; and are expressed in the animal economy, or the nice adaptation of the parts or organs of living creatures to the service they perform. There are many examples of economy in man; but the nice adaptation of the alimentary tract to the processes of digestion and absorption in proper nourishment is undoubtedly the most essential consideration in the maintenance of a harmonious and healthy body.

Some phase of the animal economy is evidently brought into action by the dietary treatment of intestinal indigestion; for by reducing the quantity and improving the quality of the alimentary mixtures in many patients with eczema and psoriasis, a better action of the gut appears to be elicited, the amount and kind of nourishment is improved, and the patient becomes healthier. In general the treatment consists of educating the patients to refine and proportion the food properly and when these requirements have been fulfilled for a sufficient length of time, the condition of the skin improves. But this improvement is also accompanied by a change in the intestinal rate and the consistency of the feces. The intestinal rate becomes slower and the feces firmer, thus indicating an increased amount of absorption. With the change in the consistency, there is likewise a change in the form of the feces. At the beginning of treatment the dejections are soft and formless as shown in Fig. 1; later they become formed with marks and similar to Fig. 2; and finally when all of the functions of the

* This investigation was made possible through a Lowell Fund.

gut appear to be in action, they are formed in units with marks, as shown in Fig. 3. Accordingly as the alimentary mixture improves it is made subservient to bodily action, is controlled by the intestinal rate and the form of the feces, and is better suited to the animal economy.

In the first place, it may seem peculiar that there is an improvement in an unhealthy skin with a slowing of the intestinal rate, as this process is supposed to have a detrimental effect on the body. Under these circumstances however, it is necessary to be critical; and while a slowing of some mixtures may be harmful, it is not so with all. Then again, estimates of the intestinal rate have not

FIG. 1

FIG. 2

FIG. 3

FIG. 1.—The soft and formless feces.
FIG. 2.—A stool formed with marks.
FIG. 3.—The feces entirely formed in units.

been made accurately; they have generally been made from symptoms rather than by ingesting a substance and observing the number of hours it takes to pass completely through the gastro-intestinal tract. And if the intestinal contents elicit one of the normal intestinal functions—antiperistalsis—there is a reversed passage of the contents in the proximal colon, which undoubtedly makes the rate slower. In the second place, stools entirely composed of discrete masses or fecal units are generally supposed to be a sign of constipation. In practice this is also an erroneous supposition; for when the aliment passes along the canal regularly at about the same slow rate, two dejections are common daily instead of one. Furthermore, as most mammals normally deject

dung entirely in discrete masses, it is impossible to conceive that all of these animals are constipated.

These conceptions are perhaps due to an ignorance of the normal intestinal rate, to an unwarranted fear of the effects of a slowing of the intestinal rate in all cases; but more particularly to a lack of knowledge of the delicacy and complexity of the normal intestinal functions in completing the cycle of digestion and absorption. These intricate processes may be explained by a consideration of the three most essential forms of intestinal motility, as shown in Fig. 4. In the first process or rhythmic segmentation an elongated mass is seen in the ileum at A, but as it passes along it is suddenly divided by a series of constricting rings into small ovoid segments shown at B, then by a second series of constrictions each segment

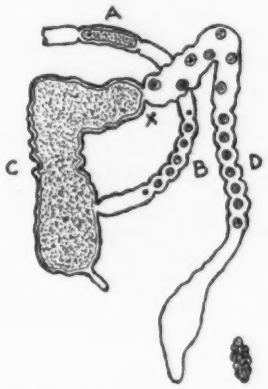


FIG. 4.—A diagram of the human gut, showing the three most essential forms of intestinal motility: (1) Rhythmic segmentation in the ileum at *A* and *B;* (2) peristalsis and antiperistalsis in the proximal colon at *C;* and (3) haustral churning in the distal colon at *D*. *X* represents the physiological constricting rings that limit the proximal colon.

will be halved, and half of number one will join with half of number two, the other half of two will join with half of number three, and so on. These segments will once more be halved and reunited. Such a division and reunion of the intestinal contents may continue for some time; and it is by this process that most of the absorption from the intestinal contents takes place. Afterward the remaining chyme is passed into the first portion of the large intestine or the proximal colon (C). Here it joins with the material already in the sac, and, being retained by the physiological constricting rings at X, is kneaded back and forth by waves of peristalsis and antiperistalsis in the second form of intestinal motility. Later the soft pultaceous material is divided again into discrete masses in the intermediate colon, and propelled along the distal colon. In

this part of the gut they are still further acted on in the final process or haustral churning at D. This action is somewhat similar to rhythmic segmentation, excepting that in this final moulding of the intestinal contents the masses remain discrete and are dejected in fecal units.[2 3 4] Such a distribution of the intestinal contents in man is shown in Fig. 5. While these complex forms of intestinal motility represent only the mechanical functions, "we may expect to find that the rate of passage through the different parts of the tube is nicely adapted to the speed of the chemical changes."[5] Accordingly, with mechanical actions and chemical changes complete, the intestinal contents must supply complete nourishment.

FIG. 5.—A roentgen-ray picture of the human colon taken twenty-five hours after a barium meal. The large shadow on the left is an outline of the proximal colon, while the distal colon is filled with fecal units. The distribution of the intestinal contents is similar to the preceding diagram.

The amount and kind of food ingested by man has been considered from many points of view, but it seems incredible to realize that these sensitive and complex functions of the gut are undoubtedly forces in the body having a selective action on the quantity and quality of the alimentary mixture; and in the requirements of the body for nourishment are far more accurate than the wisest conceptions. In other words, by selective action, mixtures that are properly refined and proportioned will complete the cycle of digestion and absorption; but those that are coarse, badly balanced or incomplete, will be passed along rapidly and cause indigestion and malnutrition. A process of this kind, whereby a

mechanical function is brought into action by a chemical stimulant is beautifully illustrated by the acid control of the pylorus. There is, however, more direct experimental evidence to support such a contention. For instance, Cannon has found that rhythmic segmentation was readily observable in cats following the action of a protein mixture, whereas a fat or carbohydrate mixture seldom elicited this complex function.[6] And Magnus found antiperistalsis present in cats with a suitable mixture, but when senna was added to the mixture this function was not elicited.[7] Then Erlanger and Hewlett observed that if dogs having most of the small intestine resected, were given proper food, they had formed feces and maintained their weight; but by adding a certain amount of fat to the food, diarrhea was produced and the animals rapidly became emaciated.[8] Finally McCarrison, by experimenting with monkeys fed on a deficient and ill-balanced diet, has found the functions of digestion, absorption and assimilation to be the first to fail, and are shown by loss of appetite, vomiting, and diarrhea.[9] From these observations it is evident that the gut has a selective action on the alimentary mixtures and passes along rapidly those that are not suitable for nourishment; but mixtures that are refined, well-proportioned and complete elicit the proper functions of the gut, are properly reduced chemically and the individual receives the nourishment in amount and kind that should be obtained from the food.

In a previous communication on "Fecal Units and Intestinal Rate, a Basis for the Study of Health and Intestinal Indigestion," the form of the feces has been presented to determine two exact conditions of the body.[10] The basis for such a hypothesis is readily understood from an examination of the specimens illustrated in Figs. 1 and 3. The former is a soft and formless mass, whereas the latter is entirely composed of units and discrete masses having a smooth surface which is broken here and there by irregular lines. These units and lines must be the result of some of the intestinal functions; but the soft and formless feces show a loss of some of these functions. It is generally considered that a loss of one of the vital functions is an indication of disease. Therefore the soft and formless stool affords a very definite means of determining a heretofore unrecognized disease—intestinal indigestion. On the other hand, the feces entirely composed of units are similar in form to the normal dung of animals. And when some of these animals are given deficient or unsuitable food, the dung often changes to a pasty and formless condition.[11] In the second place, the unit form of the feces conforms to the physiology of the gut as previously described. In the third place the requirements necessary to produce this form by man are exacting and when not fulfilled the stool changes to a soft and formless condition. In the fourth place, the

continued dejection of fecal units by an individual or race* is associated with an improved physical condition. And finally, patients whose feces have been changed from a soft and formless condition to one entirely composed of units, by a course of dietary treatment alone, have improved in health and been relieved of some very resistant conditions of disease. Therefore it is difficult not to say that the unit form of the feces is normal, and affords a very definite and immutable means of determining a heretofore unrecognized condition of the body—an improved state of well being.

If the rate at which the intestinal contents pass through the alimentary canal is nicely adapted to the speed of the chemical changes, a loss of function in one portion of the gut results in a corresponding incomplete chemical solution and insufficient or improper nourishment. Such a loss of function has been found to be due to a chemical agent, such as the ingestion of too much rich food. On the other hand the proper preparation of the food for digestion by thorough comminution and mixing with the saliva, is essential to produce the unit form of the feces. Accordingly when the food is bolted there seems to be another kind of action. This is probably bacterial in origin, and results in an excessive or peculiar putrefaction of the intestinal contents. An observation by Beaumont is quite in accord with this assertion. "Mastication is absolutely necessary to a healthy digestion. If aliment in large masses be introduced into the stomach, though the gastric juice will act on its surface, chymification will proceed so slowly that other changes will be likely to commence in its substance before it will be completely dissolved."[12] It is evident therefore that two factors are in operation in producing intestinal indigestion; one is chemical and the other a bacterial agent. Malnutrition is associated with both, but a toxemia resulting from an excessive or peculiar putrefaction of the intestinal contents is only due to bacterial action.

From experimental investigations carried on in animals, malnutrition has proved to be a factor in producing many conditions of disease. Thus McCarrison in a very comprehensive study of

* Dr. McCarrison says "For some nine years of my professional life my duties lay in a remote part of the Himalayas, where several isolated races are located far removed from the refinements of civilization. Certain of these races are of magnificent physique, preserving late in life the character of youth; they are unusually fertile, and long lived, and endowed with nervous systems of notable stability. During my association with these peoples I never saw a case of asthenic dyspepsia, of gastric or duodenal ulcer, or appendicitis, of mucous colitis or of cancer. Indeed their buoyant abdominal health has since my return to the West, provided a remarkable contrast with the dyspeptic and colonic lamentations of our highly civilized communities." R. McCarrison—Faulty Food in Relation to Gastro-intestinal Disorder, Jour. Am. Med. Assn., 1922, **78**, No. 1.

"I regret to say that I have not made observations on the form of the feces of the Himalayan tribe to which I referred, except in a most general way; but I have a distinct memory that such stools of healthy men as I have seen among these people had distinctly the unit form of health to which you refer" R. McCarrison—personal communication.

deficiency disease in pigeons, guinea-pigs and monkeys, has found that loss of appetite, vomiting, diarrhea and dysentery to be the first as well as the most pronounced symptoms and signs in these improperly fed animals. But other conditions as anemia, loss of weight, low body temperature, cardiovascular depression, asthenia, loss of hair and eczema, disturbances of the endocrine glands, headaches and symptoms referable to malnutrition of the nervous system were also produced.[13] By other investigators defective and irregularly formed teeth, scurvy, rickets, ophthalmia and neuritis have been produced experimentally in animals. One of these investigators has also alluded to the disorder of the gastro-intestinal tract with a diarrhea or "pasty feces" as the earliest and most important sign indicating a disturbance of the body.[14] The process by which one of these conditions is brought about is explained in an interesting English report.[15] "The young animals are able to grow when the fat-soluble factor is deficient in their diet, by the assumption that the animal organism normally contains reserve supplies of this factor in its own body. If this hypothesis is correct it is reasonable to assume that these reserves are mobilized for use when a deficiency occurs in the diet, but as soon as they are exhausted growth is immediately inhibited. During the period of temporary growth. . . there does not appear to be any decline in their health, but when the reserves are exhausted and the deficiency becomes felt, not only do they cease to grow but they become highly susceptible to bacterial infection. In rats this lowered resistance first becomes apparent in many cases by the appearance of a characteristic infection of the external eye, which has been provisionally classified as a xeropthalmia."

With the results of these experimentally produced conditions of disease of animals in mind, it is interesting to make a speculative survey of the human body generally dejecting the feces entirely in units, or on a unit basis and in an improved state of well being, and compare it to one with malnutrition produced by intestinal indigestion. It would seem as if properly nourished individuals acquire a certain reserve in the body, which is manifested by a strong constitution or increased degree of vital resistance. Under these circumstances if there is an acute attack of intestinal indigestion and a temporary loss of nourishment the reserve is drawn on and the tissues of the body are unaffected. But when intestinal indigestion has continued for a long time, the reserve is exhausted and the body is bankrupt; and then under the strain of continued malnutrition the weak tissue or tissues give way and some form of deficiency disease or lowered vital resistance is produced. In this study, while the skin showed the most pronounced lesions, other conditions of disease were associated with them. For instance most of the patients gave a history of dyspepsia, some were anemic, a few had asthma, and a few had arthritis. It is evident therefore

that there is one way and only one way to combat these deficiency diseases, and that is by improving the quantity and quality of nourishment to be derived from the food. With this subtle and heretofore unrecognized factor—malnutrition as a cause of some of the deficiency diseases—the treatment of eczema and psoriasis is put in a new light. But better yet, if it is possible to control the diet of children early enough, get them and keep them on a unit basis and in an improved state of well being, and then eczema, psoriasis and perhaps other conditions of disease will be prevented.

**III. Eczema and Psoriasis.** The conditions of disease considered due to a metabolic disorder are numerous; so that with a heretofore unrecognized means for the relief of intestinal disturbances, any of them might have been worked over in a preliminary study. Eczema and psoriasis have been thought to belong to this class; and lately McCarrison has been able to produce loss of hair, a rough and scaly skin, and in some cases eczema, by feeding monkeys on deficient diets.[16] Then again as they have such definite objective signs, changes in the condition of the skin are readily correlated with the form of the feces. On account of these facts these abnormal conditions afforded very good material for an initial study of intestinal indigestion as a factor in the production of disease.

In general, eczema and psoriasis have been very resistant forms of disease to treat successfully. They are often alleviated by a course of treatment, but are likely to recur. They are sometimes cured, but seldom by the same external application or dietary directions. Every now and then one is cured spontaneously or without the use of any form of treatment. But as the amount and kind of nourishment absorbed by the body undoubtedly fluctuates, and yet is in operation constantly, it is impossible to ascribe a cure to a simple remedy without taking this heretofore unrecognized factor into consideration. A history of a psoriatic illustrating some of these points is interesting to describe. The case is one of a young American, who when graduating from college noticed a few red areas on the forehead near the margin of the hair. In a few years the lesions of the scalp gradually increased in size, and similar lesions appeared on the arms and thighs. At this time he consulted an eminent dermatologist in this country, was told he had psoriasis, and was advised to use an ointment. The treatment was carried out faithfully for several months; but during this time the old lesions not only got worse, but new ones appeared. The following winter he went to Vienna, and consulted the leading skin specialist. Another kind of ointment was recommended. After the salve had been rubbed into the skin for several weeks, the lesions got paler and in a few months had disappeared entirely. The patient was very much impressed by the treatment, and procured some of the ointment to use in case the lesions

reappeared. After returning to America in about a year, new spots appeared on his body. He applied the ointment, but this time it had no effect. Thinking that it might have deteriorated he had a new lot compounded; but this too did not influence the lesions. Finally believing that the new lot might not have been properly compounded, he sent to Vienna for a fresh supply; and after using this faithfully for some time without seeing a change in the condition of his skin, he became disgusted and did nothing. Within a year, however, after he had given up all form of treatment, he noticed the red patches were becoming paler. Soon afterward many of them had disappeared, and in a few more months his skin was quite clear, and has remained so for twenty years. The course of this case and its ultimate cure is interesting, yet while one kind of treatment was used at one time, another kind at another time, and finally no treatment, the amount and kind of nourishment are the most likely factors continually in operation that could have brought about an improvement, and finally affected a cure.

The history of a case of eczema likewise expresses the influence of diet on the condition of the skin. It is one of an American boy of ten, who had had the disease since infancy. He had had many different kinds of treatment in the Out-patient Department and Skin Ward of the Hospital. At times the lesions improved somewhat; but he was unable to attend school regularly, and was generally handicapped. After using external applications and dietary treatment for intestinal indigestion, he got better and went home. In a letter from his mother the following account is given of the course of his condition. "After following the directions about diet, I found a great improvement in Buddy's skin. He felt better and went to school regularly. But on the day I was to bring him to the Hospital, I got pneumonia and of course all Bud's diet and regularity went smash. When I got well I found that his face was all broken out again, and his ears, neck and chest were red and swollen. I asked him what he had been eating; and he said apples, oranges, peanuts and candy. I put him on his diet again, also used some salve, and he is getting along finely now. Before this attack, I had just got him so that his feces were formed in units as you said they should be. It is my candid opinion that your course of treatment has done more for him than anything else, and I intend to carry it out to the best of my ability." The influence of the character of the alimentary mixture and the kind of nourishment in this case is evident; for when it was refined and wholesome the boy got on a unit basis and the skin improved; but when the diet was unrestricted, intestinal indigestion and malnutrition resulted, and these factors again produced lesions of the skin.

In this study, all of the diagnoses were made by Dr. Charles J. White, Dr. Harvey P. Towle, or other members of the Dermatological

Staff of the Massachusetts General Hospital.* In the beginning, an attempt was made to relieve intestinal indigestion in all patients with eczema and psoriasis admitted to the ward; but as the form of treatment is largely educational and some of the patients had such a low grade of intelligence, while others had so little knowledge of the English language, they were unable to understand the requirements necessary to get on a unit basis, and carry out the measures for a long period of time. Accordingly most of the patients were selected, and only those thought capable of effecting a successful outcome were worked over. The length of time the patients had had lesions of the skin varied considerably. One woman had eczema for thirty-one years; another had psoriasis for twenty-three years. The duration of most of the disturbances ranged from five to fifteen years; although there were a few cases of only a few months' standing. The forms of treatment previously tried on these chronic invalids are too numerous to review in detail. Most of them had used many kinds of salves, washes and powders, a few had tried the roentgen-ray and quartz light; some had taken arsenic, benzyl-benzoate, quinine and other drugs internally; and a few had been restricted by low protein, fat-free, and a diet according to skin reactions. Some of these measures seemed to afford relief at times. Several of the patients, however, obtained the most permanent relief by a change of environment and mode of living; for instance one psoriatic was free of lesions for a few months after spending a summer in Maine camp, and a patient with eczema likewise had a clear skin after a summer at the seashore under the supervision of a trained nurse. In obtaining a history of the habits of these patients, dietary errors were always very evident. Most of them were in the habit of eating too fast; some were continually eating between meals; a large number admitted that they ate too much sweet stuff; others spoke of taking a lot of fruit; and finally not a few had been using some form of laxative for years to relieve constipation. Those observing the feces had noticed that they were always soft. One or several of these habits were responsible for the crude or badly balanced alimentary mixture that failed to complete its cycle of digestion and absorption, and as a result of malnutrition kept the skin in an abnormal condition.

The treatment of patients with intestinal indigestion generally requires a great deal of therapeutic effort. Some having an instinctive feeling that dietary errors are responsible for the condition of the skin, understand the measures necessary to take and carry them out resolutely. They usually show an improvement in the condition of the skin after a while. Others comprehend the treatment in part or do not have the will to carry it out for a long time.

* I wish to acknowledge the kindness and coöperation the members of the Dermatological Staff have shown me in carrying out this investigation.

They are likely to improve some time. A few get well, lose control of themselves, and have another attack. And a few do not understand at all or do not consider the form of treatment sufficiently reasonable to make an effort. They continue the same or get worse. In planning a course of treatment therefore, it is not only necessary to take the disposition into account, but also the bodily condition and the action of the gastro-intestinal tract of each patient. Then again, during the course of treatment, slight alterations have to be made from time to time in the diet according to the action of the gut. That is to say, when the feces are well formed, fruit may be added to the food, but if it causes a rapidity of the rate no more should be taken for some time. The amount of food allowed of course varies with the size and occupation of the patient, but it is generally less than had been previously consumed, for it is a matter of quality rather than quantity that has to be taken into account. In this way too, a consideration of the number of calories ingested may be misleading, as it is the alimentary mixtures that complete the cycle of digestion and absorption, instead of the amount of food put into the stomach that is the principal consideration. Again the kinds of food may not be very restricted, and consist of those that are simple, wholesome, complete and suitable for the nourishment of an omnivorous animal. Candy, jam, preserves, pickles, salad dressings, ham, pork, bacon, or rich or highly spiced food should be given up for some time; cereals and soft foods should be restricted; fresh or cooked fruit should not be eaten until the intestinal rate becomes slow. Such general information while accurate may not convey an understanding of the subject; therefore a list of about the foods often suggested for a day may be helpful. Breakfast: 1 egg, 2 slices of toast and butter, 1 cup of coffee. Luncheon: 1 lamb chop, 1 portion of spaghetti and tomato, 1 piece of squash pie. Dinner: 1 slice of roast beef, 1 potato, portion of spinach, 1 roll and butter, 1 portion of rice custard. These foods amount to about 100 gm. of protein, 100 gm. of fat, and 200 gm. of carbohydrate and the accessory substances; and taken in a light breakfast and luncheon but a good dinner. The proportion should be about the same every day as a marked change is likely to affect the intestinal motility. The time of eating should be restricted to two or three meals during the twenty-four hours; and only water should be taken between meals. On a two-meal regime a little more food may be added to the breakfast given previously and eaten in the middle of the morning, and somewhat similar dinner should be allowed in the evening. This basis is valuable for obese patients and those that have had an abnormal condition of the skin for many years. All of the food must be thoroughly comminuted and mixed with the saliva. An attempt should be made to have a movement half an hour or so after breakfast and again before going to bed. In the early part

of treatment defecation is usually successful at one of these times, but if the intestinal rate is to become slower, a day is likely to pass without a dejection. If two days pass an enema or suppository should be resorted to. Later in treatment when the feces are well formed in marks or units, two dejections are common daily. When the patient is having regular dejections, estimates of the intestinal rate are of value every week or so. For this purpose a measure of 50 cc of French millet seed in half a tumbler of water is swallowed directly after an evening meal. In the beginning of treatment when the patient has soft and formless feces, the seeds are invariably seen in the dejection the next morning, and are frequently seen for two more days; or to put the test in terms of the intestinal rate, if the seed were taken Monday at 7 P.M. and were first seen on Tuesday at 9 A.M. the initial rate is fourteen hours, and then if they are seen again on Wednesday and last seen on Thursday at 9 A.M. the final rate is sixty-two hours. Later, however, when the patient is generally on a unit basis, seeds taken Monday night at 7 P.M. are not seen until Thursday at 9 A.M. or with an initial rate of sixty-two hours, and appear on Friday and Saturday, and are last seen on Sunday at 9 A.M. a final rate of one hundred and thirty-four hours. Muscular exercise is a valuable adjunct in carrying out the treatment. And by having the patient keep a record for a week every now and then of the food ingested, the time taken for the meals, the form of the feces, and finally a test of the intestinal rate, the alimentary mixtures are better controlled. A change in the intestinal rate and the form of the feces should be brought about by this form of treatment. The change may take place in months rather than weeks. But if a change is not brought about in a reasonable time, something is wrong with the alimentary mixture and further adjustment is necessary.

For two years, while this investigation has been carried on, an attempt has been made to relieve intestinal indigestion in 30 patients with eczema and 30 with psoriasis. A few of each group having kindly consented to serve as controls have had dietary treatment only and are well; but a majority of the patients have also had external washes and ointments. The results obtained with eczema are shown in Table I.

TABLE I.—RESULTS OBTAINED WITH ECZEMA.

| No. cases. | Treatment carried out. | Form of feces. | Bodily condition. |
|---|---|---|---|
| 10 | Generally | In units | Well. |
| 4 | Generally | Not known | Well. |
| 6 | Sometimes | With marks | Improved. |
| 1 | Sometimes | Not known | Improved. |
| 4 | Not at all | Soft and formless | Eczema. |
| 3 | Not at all | Not known | Eczema. |
| 2 | Not heard from | | |

The results obtained with psoriasis are shown in Table II.

TABLE II.—RESULTS OBTAINED WITH PSORIASIS.

| No. cases. | Treatment carried out. | Form of feces. | Bodily condition. |
|---|---|---|---|
| 15 | Generally | In units | Well. |
| 8 | Sometimes | With marks | Improved. |
| 1 | Sometimes | Not known | Improved. |
| 4 | Not at all | Soft and formless | Psoriasis. |
| 2 | Not heard from | | |

A glance at the results obtained in the treatment of eczema and psoriasis shows that they are dependent on the amount and kind of nourishment obtained from the food. For the patients that generally carried out the treatment got on a unit basis, and after a while have been relieved of an unhealthy skin by getting into an improved state of well being. Those that have sometimes or in part taken up the treatment, have been partially relieved of intestinal indigestion and are better. While those that have not taken up the treatment at all continue to have intestinal indigestion, malnutrition, and eczema and psoriasis. Unlike other forms, the treatment is always available for those continuing to have intestinal indigestion; and several patients have made two and even three attempts to get into an improved state of well being. A few of the patients with eczema and asthma or psoriasis and arthritis have made an improvement in both conditions of disease; but the number of cases is not sufficient to be very significant. All of the patients with constipation found, after improving the alimentary mixture and getting a slower intestinal rate, that laxatives were unnecessary, as they had one and sometimes two movements a day. The weight of many of the spare patients has increased; whereas those that were obese have lost weight. And without exception all of the patients generally on a unit basis have felt in an improved state of well being.

At this time let us stop for a moment and reflect. Think of the waste of nourishment! Think of the waste of the tissues! Think of the years and years of misery that have fallen to the lot of many of these patients! And yet there are children at the present time, who will undoubtedly follow the same course. Should we not attempt to prevent such extravagance and suffering? From studying the action of the gut of a few children, it is not a difficult matter to control the food, so that they are generally on a unit basis. In Fig. 6 is shown the feces of an infant at eleven months entirely composed of units. For three years now, this child has been generally dejecting this form of the feces, and will continue to be kept in an improved state of well being as long as the diet is carefully supervised. Other children can be maintained in the same healthy condition, and in this way the needless waste of

nourishment, the needless waste of the tissues, the needless misery, and the wholly unnecessary conditions of disease—eczema and psoriasis—will be prevented.

FIG. 6.—Feces of an infant, aged eleven months, entirely composed of units.

**Conclusion.** It is evident that when the alimentary mixture is properly refined and proportioned it is made subservient to bodily action, is controlled by the intestinal rate and the form of the feces, and is better suited to the animal economy. In this way the form of the feces affords a means of determining two heretofore unrecognized conditions of the body. One, intestinal indigestion as shown by the soft and formless stool, is associated with a disorder of metabolism and malnutrition, and may be the cause of various secondary conditions of disease. It is undoubtedly a factor in the production of eczema and psoriasis; for they have been entirely or partially relieved when the nourishment of the body has been improved; and have returned again with intestinal indigestion and malnutrition. The other, an improved state of well being, determined by the unit form of the feces, is a condition in which the aliment completes its cycle of digestion and absorption, and the individual evidently receives the nourishment in amount and kind that should be obtained from the food. At this time there are many children with intestinal indigestion soon to be crippled with some bodily disorder by this form of malnutrition. With this heretofore unrecognized factor in the production of disease, we should attempt to apply this hygienic form of treatment in the relief of intestinal indigestion as early as possible, to prevent eczema and psoriasis and perhaps other conditions of disease, and to maintain the body in a harmonious and healthy state.

**IV. Illustrative Cases—Eczema.** CASE I.—P. W. O'N., a school boy, aged sixteen years, has had eczema since infancy. Twice

during this period the skin has shown a marked improvement. He was in the ward of the hospital ten years ago for several weeks. Ointments, washes, internal remedies and various diets have been tried. Up to two years ago he used to eat fast, between meals, and a lot of candy and sweet stuff, and lately has been eating a good deal of fruit. Movements have been regular daily, but have always been soft.

The entire surface of the body is covered with lesions, but the skin of the forehead burns and smarts and is especially aggravating. The palms of the hands are much thickened, and some of the fingers are cracked. Feces soft and formless.

November 6, 1920. Put on a diet about as follows:

*Breakfast:* Egg, 2 pieces of toast, cup of coffee.

*Dinner:* Slice of beef, potato, portion of beets, slice of bread and butter, portion of ice cream.

*Supper:* Bowl of lamb stew with vegetables, 2 pieces of bread and butter, 2 molasses cookies.

November 10, 1920. Feces more formed.

November 23, 1920. Movements formless again; too much sweet stuff.

November 27, 1920. Skin improving. Initial rate twenty hours.

February 19, 1921. Skin better. Feces formed with marks and units.

April 30, 1921. Is now generally on a unit basis.

June 23, 1921. Skin shows a little improvement. Rate forty-seven hours.

October 11, 1921. Continues on unit basis; rate forty-eight to eighty-six hours.

March 30, 1922. Has jaundice.

May 2, 1922. Skin improving slowly. Face not so rough. Thick areas on palms smaller. Generally on a unit basis. Rate sixty-two to one hundred and forty-seven hours.

July 10, 1922. Skin continues to improve. Rate fifty to ninety-eight hours.

At present he is eating about as follows:

*Breakfast:* Scrambled egg and bacon, saucer of oatmeal and milk, cup of coffee.

*Lunch:* Slice of beef, potato, portion of stewed tomatoes, 2 slices of bread and butter, portion of rice pudding.

*Dinner:* Portion of chicken pie with potato and vegetables, 2 rolls and butter, cup of tea, glass of milk, portion of apple sauce, 2 pieces of cake.

CASE II.—H. L., an English Jew, aged forty-five years, and employed recently as an automobile-top maker, he has had eczema for thirteen years. The condition of the skin has been called

psoriasis, but is now a dermatitis exfoliativa. At times there was a little improvement, but he has never been entirely free of lesions. For nine weeks has had an acute attack. As a child had a few

FIG. 7

FIG. 9

FIG. 8

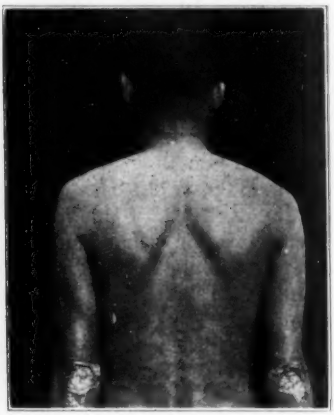

FIG. 10

FIG. 7.—Chest of H. L., with dermatitis enfoliativa.
FIG. 8.—Back of H. L., with dermatitis exfoliativa.
FIG. 9.—Chest of H. L., after four months of dietary treatment; only a little salve was used at first.
FIG. 10.—Back of H. L., after four months' dietary treatment.

attacks of eczema. Had "rheumatism" fifteen years ago; other wise general health has been good.

Always ate rapidly; spending less than five minutes at a meal, but not between meals. Used to eat a good deal of sweet stuff.

Bowels moved daily as a general rule; but sometimes had diarrhea accompanied with abdominal pain, nausea and gas. Movements are soft.

Entire body red and cracked, and covered with scales, see Figs. 7 and 8.

June 19, 1922. Given directions about diet.

June 24, 1922. Has been eating slower, spending about twenty minutes at a meal. Feces continue formless.

July 1, 1922. Feces often contain a few units.

July 8, 1922. Skin improving a little. Has been entirely on a unit basis this week. Intestinal rate seventy-four to one hundred and forty-one hours.

July 11, 1922. Went home.

October 6, 1922. Seen in Out-patient Department. Only a few lesions remain on hands and arms, as shown in Figs. 9 and 10, otherwise skin is perfectly clear. A remarkable growth of hair has appeared on the forehead. Does not have any pain in stomach, nausea or gas. Feces have been entirely formed in units since leaving the hospital.

CASE III.—A. L., a Jewess, aged fourteen years, has had eczema since infancy. At times the condition of the skin improved; supposedly according to some of the external applications and other treatments she has tried. She has been in the ward of the hospital twice, and has been coming to the Out-patient Department for treatment off and on for six to seven years.

Always ate rapidly and a lot of food; also ate between meals and a good deal of candy and fruit. Usually had a movement of the bowels regularly in the early afternoon.

April 13, 1921. At this time most of her body is covered with lesions. The patient was given dietary directions for the treatment of intestinal indigestion, and salves.

April 14, 1921. Feces are soft and formless.

April 15, 1921. No movement.

April 17, 1921. Feces contain a few units. Skin improving.

April 20, 1921. Feces are soft and formless, and due to too much fruit.

April 26, 1921. Initial intestinal rate is fifteen hours.

May 7, 1921. Skin continues to improve.

June 11, 1921. Has been entirely on unit basis for last few weeks. Skin almost clear of lesions.

July 23, 1921. The skin is now quite clear. Cautioned about eating too much fruit.

November 19, 1921. Skin is very dry. Feces have been soft and formless lately from eating too much fruit.

January 10, 1922. Has scabies and is given local treatment.

May 13, 1922. Continues on unit basis, and the skin is clear.

CASE IV.—F. L., Jewish widow, aged sixty years, has had eczema for thirty-one years. During this time she has been in the ward of the hospital seven or eight times for a few months; and has been coming to the Out-patient Department almost every week. Has tried washes, salves, arsenic, diets, Alpine lamps and roentgen-ray.

Used to eat rapidly and between meals, and also a good deal of fruit. Has been taking physic for constipation for many years. Face, hands and arms covered with red, raised itching lesions.

January 22, 1921. Given directions about diet, and put on two meals.

January 29, 1921. Bowels have been a little irregular.

February 5, 1921. Has two soft and formless movements a day without physic. Is eating too much fruit.

February 26, 1921. Skin improving a little.

March 12, 1921. Feces often formed in units.

April 23, 1921. Skin shows a decided improvement.

July 27, 1921. Is regularly on a unit basis. Skin clear. Given advice about returning to three meals daily.

September 19, 1921. Face and hands broken out with a few red patches, and there is a question of epidermophytosis. Ate something that disagreed with her and had diarrhea for a week. Permanganate put on hands, but nothing on face. Put back on two meals.

September 24, 1921. Has been back on unit basis this week; face clear and hands are better.

January 26, 1922. Skin continues clear and feels well. Still has only two meals; but generally two movements entirely in units.

CASE V.—M. C. S., a school girl, aged sixteen years, has had eczema and asthma since infancy. During this time she has been in the ward of the hospital thirteen times for three to four months at a time; and while at home had been coming to the Out-patient Department every week. All kinds of ointments and washes had been used. She had also tried fat-free and low-protein diets; and in skin tests had been found sensitive to milk, eggs, and to horse hair. One summer at seashore under the supervision of a trained nurse she got well; but lesions returned after a month at home.

Always ate rapidly, between meals, and a lot of candy and sweet stuff. Mother says her food did her no good. Stools always soft and moved two or three times a day. At this time, neck, arms and legs were covered with lesions.

August 23, 1920. The patient was told to eat only at meal times, to eat slowly, and avoid candy, rich food, and fresh and cooked fruit for the present.

September 3, 1920. Feces formed in units.

September 11, 1920. Stools soft and formless and contain large uncomminuted lumps. Eating too fast. Is applying vaseline externally.

October 30, 1920. Has been on unit basis for some time. Skin improving.

February 19, 1921. Skin continues to improve. Is regularly on unit basis and having two movements a day.

March 1, 1921. Is in the medical ward with pneumonia. Skin worse.

April 16, 1921. Skin no better. Movements continue soft and formless. Has been eating too much fruit.

July 28, 1921. Has generally been on a unit basis for sometime. Skin of neck and face normal; and hands and legs are better. Asthma also improved.

September 19, 1921. Skin continues to improve. Intestinal rate thirty-eight to one hundred and forty-five hours.

**Illustrative Cases—Psoriasis.** CASE VI.—F. J. R., an American pharmacist, aged thirty-three years, had had psoriasis for eight years. The second summer it almost disappeared. Being a pharmacist he has tried a great many internal and external remedies, but did not find one that brought an improvement in his skin.

Always ate moderately fast, and a good deal of candy and peanuts between meals. Liked fruit and ate a lot of it. Has been constipated for years and has had to take a laxative almost every night. Stools always soft and formless.

Was told to eat about as follows:

*Breakfast:* A portion of creamed beef, 2 slices of toast, cup of coffee.

*Dinner:* Portion of lamb, potato, portion of cabbage, slice of bread and butter, portion of tapioca pudding.

*Supper:* Bowl of beef soup, portion of macaroni, slice of bread and butter, piece of cake, cup of cocoa.

April 4, 1922. Movements have been irregular.

April 8, 1922. Feces sometimes formed with marks. Rate twenty-seven to one hundred and eighteen hours.

April 15, 1922. Skin improving. Movements are sometimes in units. Rate fifty to one hundred and twenty-three hours.

April 22, 1922. Generally has two movements a day entirely in units.

May 15, 1922. Since returning home has been eating too fast and too much. Had indigestion and did not feel well. A few new spots appeared on his skin. Feces often soft, with a rate of twenty-seven to eighty-six hours.

July 5, 1922. Skin improving.

July 10, 1922. Lesions have disappeared. Regularly on a unit basis and generally having two movements a day. Rate sixty-two to one hundred and thirty-six hours.

At present is eating about as follows:

*Breakfast:* Two pieces of toast, glass of milk, cup of coffee.

*Dinner:* Piece of boiled pork, potato, portion of cauliflower, slice of bread and butter, piece of apple pie, glass of milk.

*Supper:* Portion of cheese, potato, slice of bread and butter, portion of custard, glass of milk, cup of tea.

CASE VII.—J. F. C., an American laborer has had psoriasis for six months. Always ate a great deal, between meals and fast; also ate a large amount of fruit, but candy and rich food in moderation. Sometimes had diarrhea but usually had a large soft movement in the afternoon. Seldom took physic. On the advice of local doctor has been eating less and eating slower.

On examination the entire body is moderately covered with ringed raised red and scaly lesions, see Fig. 11. Feces partly formed in units, with an intestinal rate of twenty-four hours.

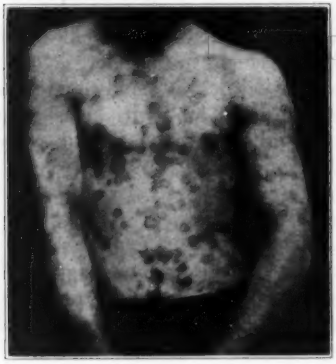

FIG. 11

FIG. 12

FIG. 11.—Chest of J. F. C., with psoriasis.
FIG. 12.—Chest of J. F. C., after three months of dietary treatment.

The patient was given ordinary house diet, but without fresh or cooked fruit, ham or bacon, and cereals. He is also using ointment.

July 6, 1921. Has had movements daily, and some of them have been formed entirely in units. Intestinal rate seventy-two to ninety-six hours.

July 12, 1921. Allowed a banana.

July 13, 1921. Feces soft and formless.

July 14, 1921. No movement.

July 15, 1921. On unit basis again.

August 13, 1921. Continues on unit basis. Skin shows a decided improvement.

August 20, 1921. Ate a banana without changing form of feces.

October 3, 1921. Skin about clear (see Fig. 12) and he eats about what he likes. Still on unit basis. Rate sixty-four hours.

December 4, 1921. Has been eating too much fruit. Feces often soft and formless. A few new lesions have appeared on chest.

January 1, 1922. A lot of new lesions have appeared. Got back to his former habits again; and has been eating too fast and too much, between meals and a good deal of fruit and sweet stuff. Feces continue soft and formless. Intestinal rate seventeen to forty-two hours. Given dietary directions.

January 29, 1922. Is back on unit basis again. Rate forty-three to one hundred and fifteen hours.

February 12, 1922. Lesions becoming a lighter color. Regularly on a unit basis. Rate forty-four to one hundred and thirteen hours.

March 27, 1922. Continues on unit basis. Rate forty-five to ninety-six hours.

April 30, 1922. Large clear areas in center of most lesions. Rate sixty-four to one hundred and thirty-nine hours.

June 3, 1922. Skin almost clear. Has not used external applications during this attack. Rate fifty to one hundred and twenty-eight hours.

August 20, 1922. The skin is now quite clear. Is generally on a unit basis. Rate sixty-two to one hundred and thirty-four hours.

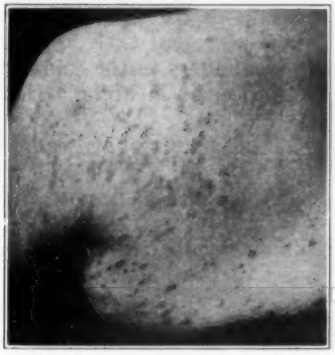

FIG. 13

FIG. 14

FIG. 13.—Shoulder of J. A. McL. with psoriasis.

FIG. 14.—Shoulder of J. A. McL. after a year of dietary treatment. This patient kindly consented to serve as a control, and did nothing but improve her nutrition.

CASE VIII.—J. A. McL., an American housewife, is moderately fat. She has had psoriasis for twenty years in patches generally covering the body; and in spite of trying various external applications and internal remedies, it has remained about the same. Has not had asthma or rheumatism.

Always ate rapidly and a large amount of food. Did not eat

between meals. Generally ate a good deal of sweet stuff, but was moderate with fruit. Took a good deal of medicine at one time. Used to have a movement of the bowels regularly on arising.

The body is generally covered with small red, raised and scaling lesions, which are larger and more numerous on lower extremities. See Fig. 13.

July 16, 1920. Given dietary directions but is not to use external or internal remedies.

July 23, 1920. Movements irregular. Has had several after breakfast that were soft and formless.

August 6, 1920. No new spots have appeared.

August 18, 1920. Intestinal rate twelve to thirty-three hours.

September 3, 1920. Feces sometimes contain a few units and generally has a movement every day. Rate twenty-four to X (not observed) hours.

September 8, 1920. Rate forty-nine to one hundred and eight hours.

September 24, 1920. Spots becoming paler. Rate fifteen to one hundred and two hours.

February 5, 1922. Letter. "Skin shows remarkable improvement. In splendid health. Movements regular and often in units. Initial rate seventy-two hours. Has lost 10 pounds."

February 27, 1922. Skin about clear. See Fig. 14.

CASE IX.—W. A., an American school boy, aged twelve years, has had psoriasis for seven years. Several times in the summer the lesions disappeared, but they returned again in winter. Has tried many ointments and salves.

Eats a large amount; is especially fond of potatoes, and eats a large number daily. Has always eaten fast, and a good deal of candy and fruit between meals. Usually has a single soft movement after breakfast.

Entire body covered with red raised scaly lesions. (See Fig. 15.) Feces soft and formless. Allowed house diet—without fruit and cereals. Used ointment.

April 5, 1921. Feces formed with marks, rate twenty-seven hours.

April 21, 1921. Has some difficulty in eating slowly. Feces soft and formless. Rate seventeen hours.

September 1, 1921. Letter. "Skin has improved a little. Stools all right and sometimes three a day. Is eating slower, but a good deal of fruit and candy."

March 17, 1922. Came to the Out-patient Department. Skin again covered with lesions. Got into bad habits once more and has been eating too much, too fast, and too much fruit and candy. Given dietary directions and ointment.

March 20, 1922. Rate fourteen to sixty-two hours.

March 25, 1922. Rate twenty-eight to ninety-six hours.

April 4, 1922. Most of the lesions have disappeared, is eating less and slowly; and no candy, little fruit. Is generally on a unit basis. Rate forty to one hundred and thirty-four hours.

October 6, 1922. Skin is clear (see Fig. 16). Is generally on a unit basis. Initial rate forty-eight hours. Has gained 3 pounds.

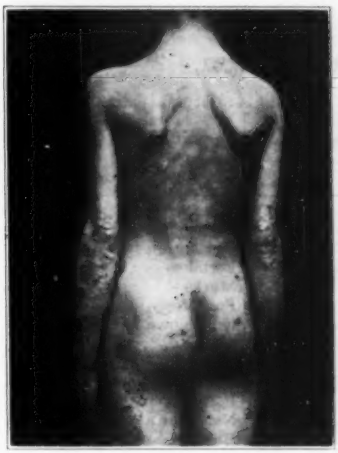

FIG. 15

FIG. 16

FIG. 15.—Back and thighs of W. A., with psoriasis.

FIG. 16.—Back and thighs of W. A., five months after generally carrying out dietary treatment.

CASE X.—O. J. B., an American housewife, is very fat, and has had psoriasis for five years. Two years ago during pregnancy it almost disappeared. Has tried all kind of external applications and diets.

Always ate fast, between meals and a lot of candy and sweet stuff. Fruit in moderation. Bowels moved any time but frequently had to use suppositories. Surface of body slightly covered with small red scaling lesions.

September 29, 1921. Given dietary directions.

October 13, 1921. No new spots have appeared. Movements more regular and better formed. Rate twelve to one hundred and eight hours.

October 20, 1921. Rate twenty-four to one hundred and thirty hours.

October 28, 1921. Rate ten to one hundred and fifty-six hours.

November 2, 1921. Rate twenty-six to one hundred and eight hours.

December 13, 1921. Skin much better. Has not used any ointment for six weeks. Movements often formed with marks. Rate nineteen to one hundred and thirty-four hours.

January 1, 1922. Rate twenty-two to one hundred and seven hours.

February 3, 1922. Skin about well. Only a few spots remain. Feces mostly in marks, but has seen a few units.

April 1, 1922. Rate twenty to one hundred and thirty-five hours. Is pregnant. Has lost about 30 pounds.

SUMMARY OF CASES—ECZEMA.

| No. case. | Age. | Duration of disease in years. | Time observed in months. | Treatment carried out. | Form of feces. | Bodily condition. |
|---|---|---|---|---|---|---|
| 1 F. B. | 9 | 7 | 12 | Sometimes | Not known | Improved. |
| 2 J. M. B. | 49 | 3 | 12 | Not at all | Not known | Eczema. |
| 3 I. M. B. | 50 | 5 | 6 | Not at all | Formless | Eczema. |
| 4 W. J. B. | 56 | 20 | 12 | Generally | Marks | Improved. |
| 5 B. D. B. | 17 | 5 | 12 | At times | Formless | Eczema. |
| 6 A. D. | 18 | ¼ | 10 | Sometimes | Not known | Well. |
| 7 M. D. | 23 | ½ | 1 | Generally | Marks | Well. |
| 8 J. C. F. | 67 | 1 | 4 | Generally | Marks | Well. |
| 9 C. M. G. | 73 | 20 | 15 | Generally | Units and marks | Well. |
| 10 E. C. G. | 16 | 2 | 12 | Not at all | Formless | Eczema. |
| 11 J. A. H. | 48 | 20 | 6 | Generally | Not known | Well. |
| 12 D. J. H. | 23 | ¼ | 6 | Generally | Units and marks | Well. |
| 13 F. T. J. | 4 | 3 | 1 | Not heard from | | |
| 14 M. K. | 52 | 3 | 8 | Not at all | Not known | Eczema. |
| 15 R. K. | 35 | 7 | 12 | Not at all | Not known | Eczema. |
| 16 H. L.* (Case 1) | 45 | 13 | 5 | Generally | Units | Well. |
| 17 F. L.* (Case 2) | 60 | 31 | 20 | Generally | Units | Well. |
| 18 A. L.* (Case 3) | 14 | 8 | 18 | Generally | Units | Well. |
| 19 J. M. M. | 60 | 5 | 8 | Generally | Marks and units | Improved. |
| 20 M. H. M. | 63 | 1 | 15 | Generally | Units and marks | Well. |
| 21 M. F. M. | 14 | 1 | 6 | Generally | Not known | Well. |
| 22 L. F. M. | 33 | 30 | 6 | Generally | Units and marks | Improved. |
| 23 A. M. | 14 | 5 | 8 | Generally | Units and marks | Well. |
| 24 P. W. O'N.* (Case 4) | 15 | 15 | 24 | Generally | Units and marks | Improved. |
| 25 S. A. P. | 22 | 20 | 1 | Not heard from | | |
| 26 W. R.* (Case 5) | 10 | 9 | 18 | Generally | Units, marks and formless | Well, two attacks. |
| 27 H. A. R. | 72 | 4 | 24 | Generally | Units and marks | Improved. |
| 28 M. S. | 16 | 15 | 24 | Generally | Units and marks | Improved. |
| 29 M. V. S. | 13 | 12 | 2 | Not at all | Not known | Eczema. |
| 30 E. W. | 4 | 2 | 8 | Generally | Units and marks | Well. |

* Given more in detail under illustrative cases.

SUMMARY OF CASES—PSORIASIS.

| No. case. | Age. | Duration of disease in years. | Time observed in months. | Treatment carried out. | Form of feces. | Bodily condition. |
|---|---|---|---|---|---|---|
| 1 W. A.* (Case 6) | 12 | 7 | 20 | Carefully of late | Units and marks | Well. |
| 2 C. A. | 58 | 20 | 3 | Generally | Units and marks | Improved. |
| 3 O. J. B.* (Case 7) | 24 | 5 | 12 | Generally | Marks | Well. |
| 4 E. F. B. | 38 | 8 | 14 | Sometimes | Marks | Improved. |
| 5 R. E. C. | 10 | 1 | 6 | At times | Units, marks soft and formless | Got well had second attack. |
| 6 A. Mc | 24 | 3 | 1 | Not heard from | | |
| 7 C. L. C. | 15 | 6 | 25 | Generally | Units and marks | Well. |
| 8 J. F. C.* (Case 8) | 25 | ¼ | 25 | Generally | Units, marks soft and formless | Well, two attacks. |
| 9 L. M. C. | 42 | 23 | 24 | Sometimes | Marks and soft and formless | Improved got worse |
| 10 E. J. F. | 32 | 14 | 12 | Not at all | Soft and formless | No better. |
| 11 A. F. | 41 | 12 | 18 | Not at all | Soft and formless | Worse. |
| 12 M. T. H. | 38 | 15 | 22 | At times | Marks | Improved. |
| 13 E. G. H. | 35 | ¼ | 6 | Generally | Units | Well. |
| 14 C. J. H. | 24 | 10 | 24 | Now and then | Marks | Improved. |
| 15 B. J. | 22 | 3 | 6 | Generally | Marks | Almost well. |
| 16 E. C. J. | 26 | 8 | 25 | Sometimes | Marks | Improved. |
| 17 J. F. K. | 48 | 6 | 3 | Generally | Marks | Improved. |
| 18 F. R. K. | 33 | 15 | 20 | Now and then | Soft and formless | The same. |
| 19 J. A.McL* (Case 9) | 37 | 20 | 8 | Generally | Units | Well. |
| 20 C. M. | 21 | 9 | 24 | Sometimes | Units, soft and formless | Well two attacks. |
| 21 A. C. M. | 50 | 20 | 4 | Generally | Marks | Improved. |
| 22 E. C. N. | 16 | 10 | 20 | Sometimes | Units, soft and formless | Well two attacks. |
| 23 E. P. O. | 47 | 1 | 1 | Not heard from | | |
| 24 B. P. | 16 | 4 | 8 | Generally | Units, marks | Well. |
| 25 L. C. P. | 33 | 1 | 4 | Sometimes | Not known | Improved. |
| 26 F. J. R.* (Case 10) | 33 | 7 | 8 | Generally | Units | Well. |
| 27 M. F. S. | 19 | 9 | 6 | Generally | Units | Well. |
| 28 G. R. T. | 18 | 6 | 25 | Generally | Units | Well. |
| 29 H. I. T. | 33 | 16 | 10 | Generally | Units and formless | Well two attacks. |
| 30 M. A. W. | 40 | 10 | 4 | Generally | Units | Well. |

* Given more in detail under illustrative cases.

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# REVIEWS.

NUTRITION AND CLINICAL DIETETICS. By HERBERT S. CARTER, M.A., M.D., Assistant Clinical Professor of Medicine, Columbia University, New York; Consulting Physician to the Presbyterian Hospital, Lincoln Hospital, Skin and Cancer Hospital, New York; PAUL E. HOWE, M.A., PH.D., Associate, Rockefeller Institute for Medical Research; formerly Assistant Professor of Biological Chemistry, Columbia University, New York; Officer-in-Charge of Laboratory of Nutrition, Army Medical School, Washington, D. C.; and HOWARD H. MASON, A.B., M.D., Associate in Diseases of Children, Columbia University, New York; Visiting Physician, Children's Service, Presbyterian Hospital, New York. Third edition. Pp. 731. Philadelphia and New York: Lea & Febiger, 1923.

To one who has used the previous editions of this work, the appearance of a third edition is most welcome. The previous editions have proved extremely practical and of great value and with the enlargements and changes, such as appear in this newer edition, the book should prove even more practical and more useful. The authors have, in addition to thoroughly revising the book, rewritten the chapter on vitamins and the chapter on food requirements of children. The chapter on rickets has also been rewritten. They have also discussed fully von Pirquet's method of feeding and have added most valuable discussion on the importance of ketone-antiketone properties of foods in relation to diabetic diets. With these additions, the subject of dietetics may be said to be presented to the medical world in a manner which leaves but little to be desired. A practical work-a-day book on dietetics is particularly important on account of the tendency of many physicians to neglect this most important branch of therapeusis except in a few diseases, such as diabetes, in which the diet is of prime importance. M.

COLLECTED PAPERS OF THE MAYO CLINIC. VOL. XIV, 1922. Pp. 1394; 488 illustrations. Philadelphia: W. B. Saunders Company, 1923.

EACH year the contributions to medical literature from the Mayo Clinic seem to grow in number so rapidly that if the present plan

of compiling these contributions yearly is continued, in succeeding years undoubtedly it will be necessary to publish them as a two-volume work. The present volume contains over thirteen hundred pages and is so large as to be almost unwieldy. Aside from this criticism nothing unfavorable can be said about the work. It is well edited and well printed. The papers that go to make up the volume will be of interest to the physician interested in any phase of medicine. The report of operations, operative procedures, results of operations, etc., are, of course, in the majority, but there are a large number of medical papers as well as those devoted to the more special branches. The catholicity of the contents may be appreciated from the fact that practically every portion of the body is covered and such diverse subjects as morphea, Meniere's disease, ketogenesis, brain tumors, water intoxication and other unusual conditions are dealt with. The breadth of the contents may be judged from the fact that in the section on the alimentary tract, for example, twenty-eight articles appear. It hardly seems just to offer a review such as this but one becomes accustomed, in considering the Mayo Clinic, to deal with quantity rather than quality, and although this review has dealt almost entirely with the quantity element of the volume, it is to be stated that the quality of the presentations compares most favorably with that emanating from any other clinic or institution in this country.

M.

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Nosography in Modern Internal Medicine. By Knud Faber, M.D., Professor of Internal Medicine, University of Copenhagen. Pp. 222; 21 illustrations. New York: Paul B. Hoeber, Inc., 1923.

It has been truthfully said that no physician is qualified to excell in his profession unless he has a broad knowledge of the history of medicine. The internist has never had a volume devoted entirely to the history of his branch of medicine until the appearance of this present volume by Faber. Although the title of the book is *Nosography*, nevertheless the title, to a certain extent, belies the true character of the work, as it is really a somewhat sketchy but extremely interesting history of internal medicine. The author takes for his theme the idea that the clinician cannot properly functionate without the ability to label every disorder and to place it within some morbid category. With this as a basis for his work he proceeds to tell us when and how various diseases with which we are now acquainted were first described as well as how other modern, present-time procedures in diagnosis have been developed. The book is a most delightful one; and certainly, as Dr. Cole has said in his introduction, the clinicians in this country are especially fortunate that Professor Faber has been willing to translate this book into the English for our benefit. M.

RECOVERY RECORD FOR USE IN TUBERCULOSIS. By GERALD B. WEBB, M.D., Consulting Physician, Cragmor, Glockner and Sunnyrest Sanatoria; and CHARLES T. RYDER, M.D., Cragmor and Glockner Sanatoria. Pp. 78; 108 illustrations. New York: Paul B. Hoeber, Inc., 1923.

IN this delightful presentation of a rather uninteresting subject, the authors have laid down rules for the care of the tuberculous individual who is on the high road to recovery, which the patient himself should read and learn. The last half of the volume is composed of special charts which the patient keeps to show the physician on his rounds. Above and below each chart is an aphorism from some well-known author which adds interest to the work and gives knowledge to the patient. The admirable features of this book are extremely valuable both for the patient and the physician. M.

MEDICAL AXIOMS, APHORISMS AND CLINICAL MEMORANDA. By J. A. LINDSAY, M.A., M.D., F.R.C.P., Professor of Medicine in the Queen's University of Belfast; Consulting Physician to the Royal Victoria Hospital, Belfast, and to the Ulster Eye, Ear and Throat Hospital. Pp. 192. New York: Paul B. Hoeber, Inc., 1923.

THE axioms and aphorisms that the author presents to us are the fruit of many years of study of medical cases. They are good practical points which the average doctor would do well to tuck away in the corner of his brain for use when various contingencies arise in his practice. M.

INDUSTRIAL HYGIENE AND MEDICINE. By E. W. HOPE, O.B.E., M.D., D.Sc., Medical Officer of Health for the City and Port of Liverpool; Professor in Public Health, University of Liverpool. In collaboration with W. HANNA, M.A., M.D., D.P.H., and C. O. STALLYBRASS, M.D., D.P.H., Assistant Medical Officers of Health for the City and Port of Liverpool, and Lecturers in Public Health Subjects, etc., University of Liverpool. Pp. 766; 123 illustrations. New York: William Wood & Co., 1923.

THIS book brings up to date, in brief and convenient form, the important facts in our knowledge of occupational health hazards, their effects and the methods of overcoming them. While somewhat less pretentious and comprehensive, it presents in the main the subject from the British point of view as did Kober and

Hanson's "Diseases of Occupation and Vocational Hygiene" from the American standpoint. It has an advantage over the latter in that includes much valuable data that has become available since the termination of the war. It is well written, fairly well illustrated and will be of distinct value to all those, medical and others, interested in the subject of industrial hygiene. M.

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VOLUME JUBILAIRE (PUBLIÉ À L'OCCASION DU CENTENAIRE DE LA SOCIÉTÉ). SOCIÉTÉ ROYALE DES SCIENCES MEDICALES ET NATURELLES DE BRUXELLES. Pp. 766; numerous illustrations. Bruxelles: Lamertin, 1922.

THIS stately volume bears strong witness to the flourishing condition of Belgian medical and natural sciences. Following an eighty-page history of the society are more than fifty scientific articles, among whose authors are Stienon, Wybauw, Bordet, Dépage and Zunz. The format of the book is excellent and the illustrations especially noteworthy. K.

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THE DEBT OF MEDICINE TO THE FINE ARTS. By J. A. NIXON, C.M.G., M.D. (CANTAB), F.R.C.P., Physician to the Bristol Royal Infirmary; Consulting Physician to Southmead Infirmary. Pp. 29; 11 illustrations. Bristol: J. W. Arrowsmith, Ltd., 1923.

THIS essay is a most interesting contribution to the correlation of medicine with the fine arts. It is nicely illustrated and well presented. M.

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MEDICAL STATE BOARD QUESTIONS AND ANSWERS. By R. MAX GOEPP, M.D., Professor of Clinical Medicine at the Philadelphia Polyclinic; Assistant Professor of Clinical Medicine, Jefferson Medical College. Fifth edition. Pp. 731. Philadelphia and London: W. B. Saunders Company, 1923.

THOROUGH revision, with an equal number of additions and omissions, has brought this edition up-to-date, without adding to its size. It will doubtless continue to be as valuable to the candidate for licensure as it has in the past. Though the answers are necessarily condensed and often incomplete, the wide range and full index make it a useful, handy book, even for some of those who have weathered that rocky point. K.

LEGAL MEDICINE AND TOXICOLOGY. Edited by FREDERICK PETERSON, M.D., LL.D., formerly Professor of Psychiatry, Columbia University; Consulting Alienist to Bellevue Hospital, New York; WALTER S. HAINES, A.M., M.D., late Professor of Chemistry, Materia Medica and Toxicology in Rush Medical College; and RALPH W. WEBSTER, M.D., PH.D., Assistant Professor of Medical Jurisprudence in Rush Medical College. Second edition. Pp. 2242; 334 illustrations and 9 plates. Philadelphia and London: W. B. Saunders Company, 1923.

ALTHOUGH especially fitted for medico-legal purposes, yet this work is interesting and valuable to the practitioner as a reference book on many subjects. Psychiatry, for example, is here more satisfactorily dealt with than in many text-books devoted solely to this subject. Of especial interest is the discussion in the first volume of the physician's legal responsibility to patients receiving gratuitous treatment, of the conditions under which a physician may refuse or discontinue treatment, of the status of insane patients and of the physician's deportment on the witness stand. Almost the entire second volume is devoted to toxicology, and the subject is covered in a clear and comprehensive manner. This, the second edition, contains a number of articles by new contributors, and many of the articles in the first edition have been revised. Among the collaborators such names as Pearce Bailey, Da Costa, E. P. Davis, Dorland, Jelliffe, Allen J. Smith and Victor C. Vaughan speak sufficiently for the authoritative character of the work.

A.

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DISEASES OF THE SKIN. By ROBERT W. MACKENNA, M.A., M.D., B.CH. (Edin.), Honorary Dermatologist, Royal Infirmary, Liverpool; Honorary Dermatologist, The Children's Convalescent Home, West Kirby. Pp. 451; 166 illustrations. New York: William Wood & Co., 1923.

THE author has included in his book all the ordinary skin affections, while the rarer conditions are briefly mentioned in smaller type. Attention has been paid to the recent advances in dermatology and the possible part played in the etiology of skin affections by anaphylactic shock and foreign proteins. A classification has been adopted on etiology. Where the causal factor was undetermined the cutaneous affections are classed on their clinical appearances, with the exception that certain dermatological conditions are grouped together because of their association with some particular structure of the skin. The illustrations are excellent, the text well written and the large type and good paper add to the value of the publication.

K.

THE URETHRA AND THE URETHROSCOPE. By F. CARMINOW DOBLE, Hon. Casualty Out-patient Surgeon, St. Paul's Hospital, London, England. Pp. 120; 47 illustrations (2 in color). London: Henry Frowde, Hodder & Stoughton, 1923.

AN interesting little monograph upon a greatly neglected subject and one toward which more attention should be directed. The first third of the book is descriptive of instruments, with a short concise sketch of the normal anatomy. Concerning the instruments, it is to be regretted that the Montague, Holborn, Harrison, Campbell and Powell urethroscopes are little known in this country, and possibly they point with emphasis to the fact that we are somewhat neglectful of this field of diagnosis and unappreciative of its importance. The remainder of the book covers the special pathology and treatment of the anterior and posterior urethra, with an admirable chapter of fourteen pages on the female urethra, a valuable and novel addition. R.

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SUGGESTION AND COMMON SENSE. By R. ALLAN BENNETT, M.D. (Lond.), M.R.C.P., Torbay Hospital, Torquay. Pp. 105. New York: William Wood & Co., 1922.

IN a few entertaining and readable pages the author speculates upon the mechanisms through which suggestion may influence the body. He warns against the danger of mental dependency inherent in psychoanalysis and outlines a plan of suggestion which he has found useful in treating patients afflicted with both functional and organic disease. A.

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THE MEDICAL CLINICS OF NORTH AMERICA. ANN ARBOR NUMBER. Vol. VI., No. 5, March, 1923. Pp. 273; 23 illustrations. Philadelphia: W. B. Saunders Company, 1923.

THE Medical Clinics of North America in their travels around this continent arrived in Ann Arbor in the month of March. The result has been the production of a volume by the Ann Arbor clinicians which compares most favorably with the volumes that have been produced by the clinicians elsewhere in the United States. Such excellent clinics are given by the dozen different contributors that it would seem unfair to pick out any particular clinic for special mention. M.

# PROGRESS

OF

# MEDICAL SCIENCE

## MEDICINE

UNDER THE CHARGE OF

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AND

ROGER S. MORRIS, M.D.,

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**Diagnosis of Circulatory Diseases.**—In an endeavor to obtain complete diagnoses of cardiac conditions, WILLIAM St. LAWRENCE, Chairman of the Committee on Cardiac Clinics of the Association for the Prevention and Relief of Heart Disease, has drawn up a diagnostic outline in which every diagnosis of a cardiac disorder includes groups in the etiology, structural change, disturbances in pathological physiology and the functional capacity of the heart diagnosis. The diagnostic scheme thus outlined, in part is as follows:

*A. Etiology.* 1, Unknown; 2, "rheumatic:" *a*, acute rheumatic fever; *b*, chorea; *c*, growing pains; *d*, tonsillitis; *e*, pharyngitis; *f*, others, as purpura arythema nodosum, etc.; 3, syphilitic; 4, bacterial (specify bacterium if possible); 5, thyroid; 6, toxic: *a*, bacterial; *b*, mineral; *c*, vegetable (specify if possible); 7, neurogenic; 8, general systemic disease (specify the disease), *e. g.*, general arteriosclerosis; 9, traumatic.

*B. Structure.* 1, Atrophy of heart; 2, enlargement of heart; 3, hypertrophy of heart; 4, cardiac infarction; 5, rupture of heart; 6, cardiac thrombosis: *a*, auricle; *b*, ventricle; 7, myocarditis, acute; 8, myocarditis, chronic fibrous; 9, fatty infiltration; 10, fatty degeneration; 11, tumor of heart; 12, endocarditis: *a*, acute; *b*, chronic; 13, cardiac valvular disease: *a*, aortic insufficiency; *b*, aortic stensois; *c*, mitral insufficiency; *d*, mitral stenosis; *e*, pulmonic stenosis; *f*, tricuspid stenosis; 14, congenital abnormality (specify lesion if possible); 15, pericarditis: *a*, acute; *b*, fibrinous; *c*, with effusion; *d*, pneumopericardium; 16, adherent pericardium; 17, pericardial effusion; 18, tumor of pericardium; 19, aortitis: *a*, without dilation; *b*, with dilation; 20, aneurysm of (specify affected vessel or heart); 21, embolism of (specify affected vessel); 22, thrombosis of (specify affected vessel); 23, arteriosclerosis, general; 24,

arteriosclerosis of (specify locality as, coronary arteries cerebral arteries, etc.); 25, arteries of (specify locality); 26, periarteritis of (specify locality); 27, no circulatory disease; 28, no cardiac disease; 29, undiagnosed.

*C. Pathological Physiology.* 1, Regular sinus rhythm; 2, vagal arrhythmia: *a*, sinus arrhythmia; *b*, sino-auricular block; *c*, simple bradycardia; *d*, ventricular escape; 3, premature contraction: *a*, auricular; *b*, nodal; *c*, ventricular; 4, simple tachycardia; 5, paroxysmal tachycardia: *a*, auricular; *b*, nodal; *c*, ventricular; 6, auricular flutter: *a*, paroxysmal; *b*, chronic; 7, auricular fibrillation: *a*, paroxysmal; *b*, chronic; 8, auriculo-ventricular heart-block: *a*, partial block, (1) prolonged conduction time, (2) dropped beats; *b*, complete block; 9, intraventricular block; 10, neurocirculatory asthenia; 11, valvular incompetency: *a*, mitral insufficiency; *b*, tricuspid insufficiency; *c*, pulmonic insufficiency; *d*, aortic insufficiency; 12, hypertension.

*D. Functional Capacity.* 1, Patients with organic disease, but able to carry on ordinary physical activity; 2, patients with organic disease, but unable to carry on ordinary physical activity: *a*, activity slightly limited; *b*, activity greatly limited; 3, patients with organic disease, but unable to carry on any physical activity, *i. e.*, who must remain in bed or in a chair.

*E. Patients with Abnormal Signs and Symptoms.* Not believed to be due to organic disease; this diagnosis to be "not cardiac—Class E."

*F. Potential Disease.* Patients without circulatory disease, but whom it is advisable to follow because of the presence or history of an etiologic factor.

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**Xanthoma Diabeticorum.**—Nicholson (*Clifton Med. Bull.*, 1923, **9**, 12) briefly reviews the literature, noting that the text-books on dermatology have articles on this condition while internists have apparently paid less attention to it. The case described is given in detail. A young man after symptoms of diabetes for a year developed small yellowish papular lesions especially over the extremities. On a strict diabetic regimen the skin lesion disappeared but due to lax adherence to diet when not under direct surveillance the diabetic condition grew worse and he succumbed in about sixteen months after appearance of the xanthomata. There was a marked upset in both sugar and fat metabolism, there being as much as 0.344 per cent blood sugar and 7 per cent blood fat at one time. There was some tendency for the rash to disappear as the blood fat decreased and to reappear as the fat increased.

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**Quantitative and Qualitative Changes in the Island of Langerhans in Diabetes Mellitus.**—Conroy (*Jour. Metabol. Res.*, 1922, **2**, 367) made an exhaustive study of multiple sections from serial blocks through the whole pancreas in 12 cases of diabetes and 12 controls of similar age, and a less thorough study of 95 routine autopsies of non-diabetics. In the diabetic group there was a marked reduction in the amount of insular tissue. The average number of islands per cross section was 73.6 as compared with 183.6 in the control series. Every case of diabetes showed more or less qualitative changes in the islands,

chiefly hyalinization and fibrosis, the latter lesion being quite widespread in some cases. A less marked hyalinization of the islands was seen in only 2 cases of the non-diabetic group. Changes involving the acinar tissue, particularly interacinar and interlobar fibrosis were seen in both series but were more marked in the diabetic series. He concludes that both quantitative and qualitative changes were operative in leading to a functional insufficiency of the insular tissue in diabetes.

**Studies on the Relation of Tetanus Bacilli in the Digestive Tract to Tetanus Antitoxin in the Blood.**—TENBROECK and BAUER (*Jour. Exp. Med.*, 1923, **37**, 479) determined the amount of tetanus antitoxin in the blood of individuals harboring tetanus bacilli in their intestinal tract and of those whose feces contained none of these organisms. The sera of 26 individuals who showed tetanus bacilli in their feces all contained appreciable amounts of antitoxin while in only two of the sera of 30 tetanus-free individuals could any antitoxin be demonstrated. In a previous paper (*Jour. Exp. Med.*, 1922, **36**, 261) they reported the isolation of *Bacillus tetanus* from the stools of 34 per cent of 78 individuals in Peking, China, and discussed the relation of this high carrier incidence to the apparently small number of tetanus infections in that country. They now conclude that an immunity is obtained by the growth of the organisms in the intestines which protects the individual against their growth in a wound. They suggest, although not yet recommending, this as a method of immunization against tetanus where such infections are liable to be common, as in trench warfare, although the danger of these individuals as carriers would have to be considered.

## SURGERY

UNDER THE CHARGE OF

T. TURNER THOMAS, M.D.

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**Hernia Operations.**—HOGUET (*Surg., Gynec. and Obst.*, 1923, **37**, 71) says that in a series of 827 inguinal herniæ in children there was no recurrence and no death. Three years of age is the usual or routine earliest age, but certain cases were operated in the first year of life when conditions demanded it. A larger series of adult inguinal herniæ has been done with recurrence in 1.6 per cent. Four deaths were noted in 963 operations, 1 of acute yellow atrophy of the liver in a man, aged forty-five years, where chloroform had been used; 1 in a man of eight-one years from cerebral embolism. In all these cases the Bassini operation was found to be most satisfactory. Seventy-six operations were performed for undescended testicle. The operation

of choice was the Bevan operation. There were no deaths in the series and no recurrence of hernia, but the testicular results were not uniformly good at any age. Cases with undeveloped scrotum gave the poorest results. The subject of direct hernia was very interesting, for in all cases of direct hernia an indirect sac can be found. There where 17 recurrences in a series of 259 direct herniæ. The Bassini operation and the Bassini operation with rectus transplantation were used. Femoral hernia was rare, for only 85 operations, or 3.4 per cent of the whole series, were done for this condition. The so-called purse-string operation was used for this series, with but 1 recurrence. Ventral postoperative or incisional hernia is not common. Thirty-four operations were performed with 1 death from pulmonary embolus and 1 recurrence. There were only 23 cases of strangulation seen among 2468 operations.

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**Surgery of the Hepatic and Common Bile Ducts.**—W. J. Mayo (*Lancet*, 1923, **1**, 1299) says that the importance of removing all stones from the common duct cannot be overemphasized. In nearly one-third of the deaths which followed operations on the common duct in Mayo Clinic, postmortem examinations revealed that all stones had not been removed. The author suggests that some of the stones which are supposed to have reformed in the common duct are leftovers. In a period of twenty-two years, 104 patients were operated on for restoration of function between the hepatic and common duct, and the duodenum, with a hospital mortality of 15. The late mortality in all the years since operation was 19. Sixty-three of the living were followed with 45 well. Cases repaired at time of accidental injury showed no marked difficulty, for the tissues are normally pliable and if as much as 2 cm. of the ducts are lost the severed ends can be drawn together by end-to-end suture. The study showed that best results in secondary reconstruction were obtained from direct union of the stump of the hepatic duct and duodenum. Cholecystogastrostomy in malignant obstructions, whether in the head of the pancreas or the common duct, has proved a remarkably satisfactory operation. For benign disease cholecystoduodenostomy is preferable theoretically and it is usually practised in the Mayo Clinic. However, in several doubtful cases, which proved to be benign, cholecystogastrostomy has given good symptomatic results.

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**False Diverticula of the Jejunum.**—F. Helvestine (*Surg., Gynec. and Obst.*, 1923, **37**, 1) says that acquired diverticula of the jejunum occur relatively rarely. Most cases are seen in old individuals. The diverticula in the majority of cases are of the false type and occur along the mesenteric border of the intestine. Intra-abdominal pressure, caused by rectal or vesical tenesmus is, perhaps, the force causing the development of the diverticula. Local traction upon the gut by adhesion, or by mesenteric vessels when shortened by sclerosis, is probably the initial cause of the formation of these diverticula in the author's cases. Acquired diverticula in the earliest stages of formation are of the true type and may continue to develop as true diverticula. In the majority of cases muscle fibers disappear by degeneration and the diverticula develop as false diverticula.

**Differentiation between Tuberculous and Non-tuberculous Inflammation of the Epididymis.**—STEVENS (*Jour. Urol.*, 1923, **10**, 85) says that double epididymitis found on examination, slightly favors a diagnosis of tuberculosis, while prior orchidectomy or epididymectomy almost invariably means tuberculosis. A scrotal sinus of over a month's duration is probably tuberculous. Moreover, tuberculosis elsewhere in the body means genital tuberculosis in over 90 per cent of such cases. In cases of over one month's duration, the examiner has some help from the rectal examination. The older the lesion, the more does definite involvement of the prostate and vesicles point to tuberculosis; after six months this becomes a very marked factor for diagnosing tuberculosis. It must be emphasized that simple inflammatory changes of the epididymis may last as long as tuberculosis. There were 4 of the 35 non-tuberculous epididymitis cases who sought advice three years or more after onset of the swelling, and but 4 of the 74 tuberculous cases with a similarly long history in the author's series.

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**Quantitative Studies with Arsphenamine.**—KOLLS and YOUMANS (*Johns Hopkins Hosp. Bull.*, 1923, **34**, 181) say that approximately three-fourths of the arsphenamine injected leaves the blood stream in a few minutes after the completion of the injection. The remaining portion is rapidly reduced in amount, but traces may be found twenty-four hours later. The drug is stored in the liver, spleen, kidneys, lungs, cardiac and skeletal muscle. There is evidence that the alteration or excretion, or both processes perhaps, have appreciably reduced the amount of the drug three hours after injection. The liver is a more important excretory organ for arsphenamine and neoarsphenamine than the kidney. The brain shows a much lower concentration of drug in the experimental animal than does any other organ. The cerebrospinal fluid during the first twenty-four hours, if it contains the drug at all, does so only in minute concentration, which is too low to estimate with accuracy.

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**Roentgen Therapy.**—O'BRIEN (*Boston Med. and Surg. Jour.*, 1923, **189**, 1) says the destruction of the tumor tissue by means of the radiation absorbed in it is the underlying principle of the new development in roentgen-ray therapy. Roentgen-ray of long wave length produced at low voltage—the so-called soft rays—burn the skin and do not penetrate deeply. Roentgen-rays of short wave length—the so-called hard rays—penetrate deeply and do not have the same tendency to burn the skin. Continental practice has not hesitated to give intensive treatments at one sitting, lasting six, ten and twelve hours. Some in America, who have tried to copy this method, have reported excellent results as to the disappearance of the malignant growths, but the patient has also disappeared. Repeated relatively small doses of roentgen-rays are to be preferred to the so-called intensive therapy of long duration, at one sitting, because of the stimulative effect on the lymphocytes and connective tissue, with general beneficial systemic effect.

## THERAPEUTICS

UNDER THE CHARGE OF
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AND
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**The Modification of Gastric Function by Means of Drugs.**—In this paper BENNETT (*Brit. Med. Jour.*, 1923, p. 366) collects the more recent experimental work concerning substances which have been demonstrated to affect gastric function. The agents may be divided into those whose chief action is on gastric secretion and those whose action is mainly on gastric motility. Atropine is the most effective drug for diminishing gastric secretion. Its hypodermic administration is less effective than its oral exhibition. The best results follow lavage of the stomach with very dilute solutions of atropine but a less laborious and almost equally satisfactory method of administration has recently been introduced by Bennett, namely, the administration of atropine in very small doses dissolved in a very large quantity of water. The action on the gastric mucosa is as definite as the action on the pupil. By washing the stomach with weak solutions of pilocarpine, it is possible to raise the HCl secretion but the absorption of the alkaloid soon leads to salivation and if the saliva is swallowed, the acidity of the gastric juice is reduced. The group of substances that neutralize the gastric juice after it has been secreted includes the alkaline salts. There is a considerable divergence of opinion as to the exact effects of sodium bicarbonate on secretion and acidity. Bennett's experiments led him to conclude that sodium bicarbonate, given before meals, causes an actual increase in the acid secretion after the meal, while if the alkali is administered later, the acid already secreted is neutralized; a period of neutrality lasting nearly an hour is followed by a rapid rise in gastric acidity to the original level. It would therefore appear that sodium bicarbonate tends to excite the gastric mucosa to increased secretion and that this effect more than counterbalances the neutralizing effect of the salt. Other alkaline salts, especially magnesium oxide and bismuth oxycarbonate, have a less stimulating effect, and weight for weight, magnesium oxide possesses the greater neutralizing power. In hyperacidity these salts should be administered at such periods after meals as will lead to their neutralizing effects coming into play without there being any possibility of their stimulating the gastric cells. For inhibiting secretion, atropine is far more efficacious than the alkaline salts. In gastric therapy, sodium bicarbonate finds its greatest usefulness in those rather rare cases where there is an excess of mucous and a low acid secretion. Hydrochloric acid is the most valuable agent for replacing a deficient acid secretion. It must be administered in rather larger doses (2 drams of the B. P. dilute acid) than are usually

recommended. There is no unimpeachable evidence that bitters have any marked effect on gastric function; there may be subjective impressions that appetite is improved but Carlson's studies show that the amount of food ingested is not considerably increased. Gastric motility isi mpaired by atropine and emptying of the stomach is somewhat delayed (rarely by more than fifteen minutes). In pylorospasm atropine often causes marked relief. Pilocarpine, on the other hand, increases gastric movements and hastens emptying of the stomach. The effects of strychnine on gastric motility are very interesting; in doses of 0.5 mg. gastric peristalsis is at first greatly increased and emptying of the stomach is accelerated; 1 mg. doses cause an initial increase in peristalsis but at the end of thirty minutes the contraction of the pars pylorica is sufficient to delay complete emptying of the stomach; with larger doses these symptoms are exaggerated and the net result is further delay in the emptying of the stomach.

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**The Properties of Certain "Colloidal" Preparations of Metals.**—A cursory survey of the literature dealing with the therapeutic action of metals in colloidal form gives the impression that they act as specific cures for every known disease. CLARK (*Brit. Med. Jour.*, 1923, p. 273) investigated the properties of some of the more widely advertised "colloidal" preparations. Dialysis and ultrafiltration experiments indicated that oscol ferrum and argentum were true colloidal solutions and were stable on exposure to air. Ultrafiltration experiments showed collosol antimonium and arsenicum and oscol arsenicum and stibium contain a mixture of colloidal and non-colloidal metal and on exposure to air all the metal tends to pass into true solution. Very little quinine in collosol quinine is in colloidal form and no demonstrable quantity of iron in collosol ferrum appears to be in colloidal form. In diffusion experiments oscol ferrum behaved like a colloidal solution but oscol iodine and collosol ferrum acted like non-colloidal solutions. Collosol and oscol iodine apparently suffer exactly the same fate in the body as does ordinary iodine; the "colloidal" preparations contain only traces of iodine (0.025 per cent of free iodine with 0.015 per cent of combined iodine) and are therefore not likely to induce iodism. Collosol and oscol arsenic and antimony were nearly as toxic as non-colloidal solutions on intravenous injection; they were only about half as poisonous on intramuscular injection because the organic matter in them hindered their absorption. Tartar emetic was superior to either collosol antimonium or oscol stibium as a trypanocidal agent and there was no demonstrable difference in the activity of arsenious acid and xollosol arsenicum. The toxicity of the collosol preparations was about the same as that of the non-colloidal preparations of the same substances. Metals in colloidal form appear to exert only a feeble disinfectant action *in vitro* but if allowed to remain in contact with the bacteria for a prolonged period they acquire antiseptic properties because they are broken down and liberate free ions. There is no foundation for the claim that metals in colloidal form continue to circulate in the blood for a long time and act there as antiseptics. When administered intravenously colloidal metals produce a general reaction very like that following the injection of peptones or non-specific vaccines. This is a "protein shock" reaction and is due to the fact that collosol and oscol

preparations contain breakdown products of protein in considerable quantity as protective colloids. Our knowledge of the value of non-specific protein therapy is so vague that the fact that colloidal preparations may belong to this class gives little guidance to their therapeutic value.

## PEDIATRICS

UNDER THE CHARGE OF

THOMPSON S. WESTCOTT, M.D., AND ALVIN E. SIEGEL, M.D.,
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**Mongolism in One of Twins and the Etiology of Mongolism.**—HALBERTSMA (*Am. Jour. Dis. Child.*, 1923, **25**, 350) reports 5 cases, and he thinks that all of these cases of mongolism idiocy resulted from so-called two-egg pregnancies. As is known, there are two types of twins. The one-egg, or identical twins, are derived from a single egg which has formed two embryos. They are always of the same sex, are enveloped in the same chorion and there is only one placenta. The children show marked resemblance. As the entire heritable nature is common to both, germinal affections will be equally present in both, as for example epilepsy and asthma. Acquired diseases, of which the causes may be sought in the condition, such as syphilis of the mother during pregnancy, will also manifest themselves in both twins. In the two-egg, or fraternal twins, there have been two eggs, which were fertilized separately. Each embryo is enveloped in a separate chorion, and they no more resemble each other or seem more closely related than ordinary brothers and sisters, and their sex may be different or the same. One of them may be affected by an inheritable disease, such as achondroplasia, while the other may be absolutely normal. Diseases of the mother that might affect the embryo, such as syphilis, will generally manifest themselves in both twins, although the symptoms may vary. If mongolism were not germinal in origin but acquired, then the occurrence of mongolism in one of twins would be a surprise. A review of the literature revealed 15 such cases and only 2 cases of mongolism in both twins. It would be difficult to imagine how one child could remain absolutely normal while the other showed the numerous and universal symptoms of mongolism. If the mongoli m were due to defects inherent in the germ plasma the occurrence of mongolism in one of twins would be comprehensible, but it would only be possible in a two-egg pregnancy. All the cases in the literature had different sexes from the normal twins and were undoubtedly two-egg pregnancies. In the cases where both twins were mongolian idiots, it was only possible in one-egg pregnancies. It was interesting to note in the cases reported that only in two instances were the twins of the same sex.

**The Treatment of Vascular Nævi with Radium.**—RULISON and MCLEAN (*Am. Jour. Dis Child.*, 1923, **25**, 359) found that radium

therapy is expensive and time-consuming. There is also the chance of overtreatment, with subsequent ulcer formation and very tardy healing. The possibility that telangiectasia and atrophic skin may result, followed later by keratosis and epithelioma, has also been considered. The advantages of radium treatment are the freedom from pain, the very gradual changes in the lesion, which can be watched from treatment, and the probability of causing the lesion to disappear without appreciable damage to the overlying skin. In addition there is the advantage of rapid healing of ulcerations and hemorrhagic areas, the absence of scarring or when scarring is unavoidable the production of a smooth, supple and comparatively inconspicuous scar. It is possible to treat successfully lesions that because of their location are not suited for treatment by other procedures, as for example nævi of the eyelids or others on account of their depth and extent. Most potent of all reasons for using radium in treating angiomas is the fact that radium exerts a definite, specific, selective action on the abnormal endothelial cells lining the bloodvessels, which are at fault in the process. Not all angiomas respond equally well to radium treatment. The port-wine mark is very obstinate, since flat angiomas generally are more refractory than the raised. Many nævi of small size and situated on covered parts are easily destroyed by more rapid means.

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**The Schick Test.**—ZINGHER (*Am. Jour. Dis. Child.*, 1923, **25**, 392) reports the results from more than 150,000 tests. A high percentage of positive Schick reactions was found among the children of the entering classes in the kindergarten and the primary grades. This varied in different schools from 60 to 85 per cent. The toxin-antitoxin injections could be given to these children without preliminary Schick testing. The schools located in the less congested sections of the city and attended by children of the more well to do showed, as a rule, from 100 to 200 per cent more Schick positive reactions than those located in the crowded sections and attended by children of the poorer classes. Repeated exposure to infections with the diphtheria bacillus is probably the most important factor in the development of a so-called natural immunity to the disease. Racial and hereditary family factors also play a role. Girls showed a somewhat larger percentage of positive Schicks than boys. They also showed more negative pseudo- and positive-combined reactions. This also held true for adults. The high percentage of positive Schick reactions among young children of the pre-school age may, under certain conditions, render it advisable to simplify the procedure of immunization by omitting the preliminary Schick test. These children should then receive three injections of toxin-antitoxin, but a supplementary Schick test should be performed after six months to prove the immunity.

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**Outlook of Nephritis in Children.**—ASHBY (*Brit. Med. Jour.*, 1923, **1**, 408) found that the occurrence of nephritis after scarlet fever varied in different epidemics, but it may be estimated at about 10 per cent. The outlook is usually good, and it is rare for the nephritis to become chronic. The amount of albumin in the urine does not seem to alter the outlook, for a patient with a large amount may get quite well while one with only a small amount may do badly. The worst cases are

those which have remained untreated for some days and those in which suppression of urine is present, as uremia is liable to occur. The preventive treatment consists in great care during the two or three weeks after the onset of the scarlet fever, especially as regards catching cold. In cases of acute nephritis the outlook is mostly bad. They run a course of from three months to two years, but complete restoration of health does not often occur. The child may get fairly well, but he is liable to further attacks which damage the kidneys still more until he becomes waterlogged and dies. Toward the end very little urine is passed, and dyspnea becomes a marked feature, so that the patient has to be propped up in bed. Uremic convulsions are also common.

## DERMATOLOGY AND SYPHILIS

UNDER THE CHARGE OF

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**Tryparsamide in Neurosyphilis.**—LORENZ, LOEVENHART, BLECKWENN and HODGES (*Jour. Am. Med. Assn.*, 1923, **80**, 1497) report on the results of a prolonged and detailed study of the action of N-phenylglycineamide-p-arsonic acid (tryparsamide) upon neurosyphilis and particularly paretic neurosyphilis in man. The action of this drug is apparently not directly spirillicidal, but has to do rather with the resistance mechanism of the disease. It will be recalled, however, that Voegtlin and his co-workers have shown that the arsenic content of the spinal fluid after the administration of tryparsamide is higher than after the administration of any other arsenical. The technic of administration recommended by these authors consists of 3-gm. doses given weekly for a period of eight weeks, with intramuscular injections of mercury salicylate in 1-gr. doses, the latter being given weekly between the injections of tryparsamide. A rest period of from five to eight weeks between courses is given and two or three courses are employed in resistant cases. Relapse is more common when tryparsamide is used without mercury and may occur as late as two years after the institution of treatment, although the symptomatic improvement is remarkably persistent. Because of the pronounced constitutional benefits apparent during the treatment, the use of the drug is recommended in Wassermann-fast cases, in emaciated syphilitics and in those beyond middle age as well as in resistant neurosyphilis. There is a remarkable freedom from local or general reaction in the use of the drug which seems to permit its employment in cases which are reactive to other arsenicals. Stress must be laid on the fact that tryparsamide possesses the potentiality of injuring the optic tract and that it should not be used in cases showing degenerative changes in the optic nerves or retina. A careful examination of the eyes should precede any attempt to use the drug, and the development

of blurred vision during its employment is a signal for immediate cessation of the treatment. The preparation is not on the market, but is being employed by various observers under license of the Rockefeller Institute.

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**Gland Puncture in Syphilitics.**—OELZE (*Deutsch. med. Wchnschr.*, 1923, pp. 86–87) reports that he is unable to equal the 80 to 90 per cent positive puncture claimed by a number of authors. He cites Schultz, Sutton, Eberson and Mitchell, who recommend various procedures. He has adopted the following technic: (1) Disinfect the shaved area with ether, alcohol, iodine; fix the gland between the forefinger and thumb of left hand; choose a moderately indurated gland. (2) Use a large gauge needle, with short bevel. (3) Use a 1 or 2 cc syringe with 0.25 to 0.5 cc physiological sodium chloride solution. (4) The needle is attached to the syringe and advanced into the gland. (5) The needle is turned several times within the gland. (6) The saline solution is then injected. (7) The syringe is disconnected from the needle and one waits two to five minutes turning the needle several times in the meantime. (8) The needle is again connected with the syringe and the contents of the gland aspirated. (9) The syringe with its contents are withdrawn and a few drops of the material are studied in the usual manner. Strict adherence to the finest details of the technic is necessary to obtain the greatest percentage of positive punctures. The method is of special value in diagnosing puzzling lesions that had had local treatment during the period when the Wassermann reaction is still negative.

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**The Wassermann Reaction in Pregnancy.**—HINTON (*Jour. Syph.*, 1923, **7**, 155) discusses the results of the Wassermann reaction performed on the blood of 10,427 pregnant women in various Massachusetts hospitals. The method employed utilizes two cholesterinized antigens prepared from a plain alcoholic extract of human heart muscle and a more sensitive antigen also cholesterinized, made from guinea-pig hearts. Sheep's cells are used as the indicator substance. Employing this system, the author obtained 0.56 per cent positives in 3701 naval aviation students and 40.49 per cent positives in a penal group of 864 women. The proportion of positive results obtained in the 10,427 pregnant women examined was 4.18 per cent and doubtful results were obtained in 3.85 per cent. Studies of the cord blood indicate that it is only about one-third as effective in the detection of syphilis by the Wassermann test as that obtained by the usual venous puncture. Among the race groups, negroes lead the list of positives with the proportion of 33.3 per cent. Native Americans, Syrians, Portugese and Scotch stand next. The lowest percentages were among the Jews of native, Russian and Polish extraction and among the Swedes. The highest proportion of positives occurred in the age group of seventeen to twenty-two years. The author considers that a properly standardized cholesterinized antigen yields a negligible number of false positive reactions in child-bearing women.

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**Studies of the Wassermann Reaction on Syphilitic Exudates.**—STERN and RYPINS (*Jour. Lab. and Clin. Med.*, 1922, **8**, 86) have

followed up the work of KLAUDER and KOLMER (*Arch. Dermat. and Syph.*, 1922, **5**, 566) by a study of the local Wassermann reaction on the exudate from 34 demonstrated syphilitic primary lesions. In the majority of these cases, the blood Wassermann reaction was negative and secondary manifestations were absent. In all of the 34 cases, the local Wassermann reaction on the chancre exudate was positive in amounts as small as 0.0125 cc of serum. This very small amount of serum can be collected from practically any suspected lesion with a graduated capillary pipette so that the method is readily available under the conditions of ordinary practice. The surface of the lesion is sponged off with normal saline after which the lesion is dried and gently squeezed. If not enough serum is obtainable, slight scratching with the end of the glass capillary tube may be carried out, inasmuch as a small amount of blood does not affect the reaction. Local treatment of the lesions with a spirocheticide, while it may render the dark-field examination negative, does not appear to influence the local Wassermann reaction. The authors' observations appear to establish a local as well as a hematogenous site of origin for the specific complement-fixing bodies. The use of the local Wassermann is not proposed as a substitute for the dark field when this is positive, but when the dark field is not available or local treatment has been instituted the local Wassermann reaction has great value and may at times constitute the earliest means of obtaining a diagnosis.

## OBSTETRICS

UNDER THE CHARGE OF

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**A Mistaken Diagnosis and Its Result.**—SIGWART (*Zentralbl. f. Gynäk.*, 1922, **46**, 1808) describes the case of a woman, aged twenty-three years, brought to the clinic with the history of an incomplete abortion and perforation of some portion of the abdominal or pelvic viscera. The patient was in wretched condition, the abdomen painful and distended; the pulse very rapid and so weak that it was felt with difficulty. The temperature was slightly subnormal. There was a slight discharge of blood from the vagina, and there were small lacerations and bruises about the vulva. On introducing the fingers in examination, they passed on the left side through an extensive tear into the abdominal cavity, and on opening the abdomen there was partially decomposed blood, which gave, on examination, a pure culture of streptococci. There was a general peritonitis present. At the fundus and posterior portion of the uterus there were three perforations. On the left side and behind the uterus was a mass which looked at first sight like the perforated large intestine, but on closer examination proved to be an emptied ovarian cyst. On removing this from its location the most

extensive injury to the uterus was found on the left posterior portion where the uterus was torn open freely. Total extirpation was performed, and it was found that the left uterine artery had been torn, and that there had been free bleeding. The patient died fifteen hours after operation. At autopsy, septic peritonitis and endocarditis with streptococcus infection were present. The case had been diagnosed as an early pregnancy and incomplete abortion, and the physician had dilated the uterus by several attempts, which were carried out in his office. Finally, with the aid of a midwife, it was decided to complete the abortion in the lodging of the patient. The midwife had given the anesthetic. The physician had introduced the hand when fluid escaped. In his efforts to empty the uterus as he supposed, the patient had become infected and death had followed. In commenting upon the case, it is remarked that a mistake in diagnosis between early pregnancy and a small ovarian cyst may readily be made. If in early pregnancy the uterus be in an abnormal situation, or if there be inflammatory conditions present, perforation of the uterus may readily be accomplished. A perfectly clear history of these cases is never obtained; but there is one point of criticism in the management of the case upon which the writer is emphatic. The practice of dilating the uterus or performing minor operative procedures upon these patients in the office of the physician is to be strongly condemned. If the case is complicated and accurate diagnosis is not made, no procedure can be safely and efficiently carried out, and such treatment is followed by disaster to both patient and physician.

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**Double Tubal Gestation, Operation, Recovery.**—Amaral (*Gynéc. et obst.*, 1922, No. 5, p. 319) describes the case of a multipara, aged twenty-nine years, whose general health had been good. She had had four children living and in good health, and her gestation and labors had been normal. There was irregular menstruation with metrorrhagia, followed by pain in the right iliac fossa, extending to the opposite side and down the thigh. There was a pronounced general disturbance and the hemorrhage became considerable. The patient expelled a small portion of material from the uterus with clots. On internal examination there was a small tumor on the left side, and on the right the condition could not be clearly made out. At operation both tubes were pregnant, and this was proven by the microscopical examination of the tissues after the removal.

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**The Action of Morphine and Scopolamine upon the Fetus.**—Horwitz (*Lancet*, 1922, **2**, 1303) endeavors to combat the statement frequently made that morphine and scopolamine tend to produce asphyxia in the fetus. On the contrary he states that in 500 deliveries under scopolamine but 1 child died during birth. He believes that where moderate doses are given that the child does not inspire too soon, and so does not inhale amniotic fluid, vaginal epithelium and bacteria from the genital tract of the mother. In this way mortality accompanying labor is reduced.

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**Coiling and Knotting of the Umbilical Cord.**—Sellheim (*Zentralbl. f. Gynäk.*, 1922, **46**, 1746) reviews the various theories concerning the

developing of twisting, coiling and knotting of the umbilical cord. He speaks of what he calls the impulse to rotate, which originates in the pulsation of the cord, and states that the first beginnings of complications arise in the simple turning of the fetus or the umbilical cord upon its axis at its insertion. This resembles the situation which develops in an ovarian tumor or a tumor of the Fallopian tube. If the tension of the cord is somewhat lessened after this, the tendency is to develop coiling about the child, and this, he believes, is the second degree in the rotary motion. If the cord twists against the fetus or if the fetus is brought considerably against the cord, then will occur the development of coiling and knotting in proportion to the length of the cord and the conditions present.

**Fibroid Tumor; Ovarian Cyst Complicating Pregnancy.**—In *Gynécologie et obstétrique*, 1922, No. 5, pp. 456, 549, SCHOCKAERT of Louvain reports two interesting cases. His first was a Cesarean hysterectomy for pregnancy, complicated by a fibroid tumor, which was in front of the presenting fetal part, making labor absolutely impossible. It was a fibroma prævia. Two excellent illustrations show this unusual condition. The patient's convalescence was complicated by sudden failure of the secretion of milk and severe headache, but otherwise she did well. The second case was that of a woman pregnant a month, who had a cyst of the left ovary, with twisted pedicle. The diagnosis in the case was not clear, for the history was very suggestive of tubal gestation. There was good reason for opening the abdomen, and at operation the diagnosis was apparent.

**Stenosis of the Birth Canal Following Labor.**—STANCA (*Zentralbl. f. Gynäk.*, 1922, **46**, 1769) reports two cases of this rare condition. The first followed extensive lacerations of the genital tract and a radical operation for the cure of cystocele and rectocele. After an effort had been made to open the vaginal canal by dilatation, a radical operation was performed, which was followed by a practical cure. His second case was a multipara, who after a confinement, had a chronic ulcer develop in the genital tract, which was treated by a midwife with concentrated solution of sulphate of copper. This ulcerated surface slowly healed, but produced complete atresia. The third case is reported following a confinement in which extensive laceration, without adequate repair, had occurred.

**Glycemia and Glycosuria in Pregnancy and Their Diagnostic Value.**—DOSSENA (*Ann. di ostet.*, 1922, No. 10, p. 747) has made experiments to determine the diagnostic value of these conditions in pregnancy. His article is of considerable length and contains a review of the literature and description of different chemical methods employed in recognizing these substances. Glycosuria can be predicted by variations in the curve of glycemia, and such variations point to a deficiency in the function of the liver and modification of the equilibrium in the metabolism of pregnancy; and these factors are of some value in forming an idea of the integrity and functional capacity of the renal filter as regards its ability to permit the passage of sugar in solution.

# GYNECOLOGY

UNDER THE CHARGE OF

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**Carcinoma of the Cervical Stump.**—The startling figures on the incidence of cancer of the cervical stump has awakened the interest of Davis (*Boston Med. and Surg. Jour.*, 1923, **188**, 304) in the subject to such an extent that he has reviewed all cases of cancer of the cervix that have entered the wards of the Massachusetts General Hospital for the five year period preceding January, 1922. There have been 123 such cases of which 8 or 6.5 per cent were cases of cancer of the cervical stump after supravaginal hysterectomy for fibroid tumors of the uterus. In these 8 cases carcinoma in the stump was detected at the following intervals after hysterectomy, twenty-four years in 1 case, fifteen years in 1 case, five years in 1 case, two years in 3 cases and less than one year in 2 cases. It is safe to assume that in the 3 cases in which cancer was found from five to twenty-four years after the original hysterectomy, that the disease was not present at the time of the first operation but was a new development. In the 5 other cases it seems quite probable that there was co-existent carcinoma at the time of the original operation. The histories show that in 2 cases a bloody vaginal discharge was noted almost immediately after the operation, in 1 case it was noted five months, in another ten months, and in the last, one year and eight months after operation. Pathological reports on the original growth were available in 4 cases, 3 of which showed simple fibromyoma, but in spite of this, vaginal bleeding was noted almost immediately after discharge in 1 case, after five months in 1 case, while in the other the first symptom of trouble was a foul vaginal discharge without blood appearing one year and eight months after operation. Davis believes that of the 4 cases which had their original operation in the Massachusetts General Hospital, cancer was present at the time of the original operation in 2 cases but was overlooked. That the occurrence of cancer in the retained stump bears little relation to the trauma of childbirth is shown by the fact that in 3 of the 8 cases the patients were single women. The explanation is undoubtedly to be found in the fact that these were cases of overlooked co-existent adenocarcinoma of the body of the uterus. The treatment which was instituted in these 8 cases of carcinoma of the stump consisted of radical excision of the stump by laparotomy in 2 cases; in 1 other case an attempted excision was abandoned on account of adhesions and the patient referred for radium treatment. In 2 cases the growth was cauterized and then referred for radium, while in 3 cases the patients

were directly referred for radium without any preliminary operative interference. The results of treatment have not been encouraging. In the 2 cases of excision the results of treatment are unknown, but of the 5 cases referred for radium treatment, 4 died of the disease within a year and a half while the sole survivor has well-marked evidence of extensive pelvic invasion. Carcinoma develops in the cervical stump in different ways. A co-existing adenocarcinoma of the body of the uterus which is overlooked in the original operation for fibroid tumor, may be the original focus from which the cervix is secondarily invaded. Davis believes that this is the commonest cause and accounts for at least one-half of his cases. A fibroid may have undergone so-called carcinomatous degeneration at the time of operation and act as a source of secondary infection of the retained cervix, or there may be independent but co-existent carcinoma of the cervix, either of the squamous-cell or the adenomatous-cell type which is overlooked at the time of the operation. Finally carcinoma, either squamous-cell or adenomatous-cell type may develop in the cervical stump after operation as a distinct entity. Combining all these forms of carcinoma, Davis assumes that the incidence of cancer of the cervical stump following supravaginal hysterectomy for fibroid tumors is 2 to 3 per cent or more, varying with the degree of accuracy with which cases of co-existent carcinoma are detected at the time of operation and the length of time that cases are kept under observation. How should this be met by the operating surgeon? Total removal of the uterus for fibroids is theoretically logical and correct, if the mortality rate is not thereby elevated above the percentage of incidence of the disease itself. A few experienced operators doubtless may be able to accomplish this, but in the hands of the average surgeon now doing supravaginal hysterectomy, the complete operation would undoubtedly elevate the mortality considerably above this point and there would be a considerable incidence of distressing complications in the way of vesical, ureteral and rectal fistulæ. Davis suggests that in every case in which hysterectomy is contemplated in the treatment of fibroids, the cervix should be inspected and the uterine cavity curetted as a preliminary measure, any suspicious tissue should be subjected to an immediate microscopical examination before any operation is performed. If the microscope reveals cancer, a total hysterectomy should be done. If no evidence of cancer is found a supravaginal hysterectomy may be done unless the appearance of the cervix arouses a strong clinical suspicion of malignancy. Only with increasing experience and skill should total hysterectomy be adopted by surgeons generally as a routine procedure. A careful follow-up system should be adopted for all cases of incomplete hysterectomy and patients warned that the reappearance of vaginal bleeding or discharge after operation calls for prompt examination. When cancer of the retained cervix does occur, its treatment is a serious problem. Early cases may be treated by radical abdominal excision, but the operation is even more formidable than the Wertheim operation itself. Radium undoubtedly offers the best chance in the great majority of cases.

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**Value of Ovarian Extracts.**—With the intention of establishing some definite method of determining the value of the various ovarian prepa-

rations, GEIST and HARRIS (*Endocrinology*, 1923, **7**, 41) undertook an experimental investigation upon several ovarian extracts from four commercial laboratories. It is a well-established fact that after castration a definite atrophy of the uterus takes place, and it was with the idea of using this phenomenon as an indicator that the experiments were undertaken. They attempted to determine, if by the administration of these various preparations by intravenous injections, the atrophy could be prevented, minimized, or, if possible, hypertrophy could be obtained. At the same time, the breasts were also observed, as well as the thyroid, the hypophysis and the adrenals, to determine if the administration of these preparations had a definite effect upon any of them. Controls were made by subjecting animals to the same operative procedures, but without administering the drugs. The ovaries and a small portion of the tubes were removed from rabbits without interfering with the blood supply to the uterus. At the same time a small segment of the uterus near the horn was removed as a control and one breast also. Before operation the animals were weighed, and when they had recovered from the operation, after a period of usually forty-eight hours, they were put on the same food as before operation. In all cases the uterus showed atrophy, although not so marked in those killed early as in those permitted to live for two to three months. The breasts, as the uterus, showed marked atrophy. There is an actual diminution in the size of the organ, with a disappearance of the secretory gland tissue and a collapse and diminution in the caliber of the ducts. The pituitary gland did not show much change, while the thyroids of the injected cases appeared somewhat enlarged with a tendency toward the formation of small cysts in a few cases. There was an average loss of 16 per cent in body weight. Analyzing their results, they find that the injection of commercial ovarian extracts does not prevent the atrophy of the uterus following castration in rabbits, therefore there is nothing in these preparations sufficiently active that will substitute for the hormone that normally maintains the nutrition of the uterus.

## PATHOLOGY AND BACTERIOLOGY

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**The Relationship of the Orange and White Pyogenic Staphylococci with Special Reference to Vaccine Therapy.**—The value of vaccine therapy in staphylococcus infections is now generally acknowledged.

Most experienced workers deprecate the use of stock vaccines and claim that only autogenous vaccines yield good results. Inasmuch as local conditions made the use of stock vaccines for the treatment of staphylococcic infections imperative, KLIGLER and KRAUSE (*Jour. Infect. Dis.*, 1923, **32**, 133) examined thirty-five strains of staphylococci culturally and serologically with a view to determining certain type strains, from which an effective polyvalent stock vaccine could be prepared. The strains were isolated mainly from furuncles, abscesses, mastitis and acne. Although certain quantitative cultural differences existed, it was not possible on the basis of these characters alone to differentiate the pathogenic staphylococci into distinct types. The results of the serological tests performed by agglutination and absorption reactions, employing immunized rabbit serums, indicated that there was a definite group relationship between the orange and white staphylococcus, but that the aureus serums were more specific and did not agglutinate the albus strains to the extent which the serums of the latter agglutinated the aureus cocci. The absorption tests confirmed the conclusions based on the agglutination tests, namely, that the two pigment types were related. Both types of cocci produced group agglutinins for orange and white cocci, but there was a distinct difference between the two varieties—the aureus being a more homogeneous and defined type. With these facts at hand, polyvalent vaccines were made from two main types of orange and two strains of white staphylococcus. It was found that these stock vaccines were as effective therapeutically as were autogenous vaccines.

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**Immunity Studies of Rocky Mountain Spotted Fever: I. Usefulness of Immune Serum in Suppressing an Impending Infection.**—For the past twenty years Rocky Mountain spotted fever has been the subject of study by many investigators. As a result, it has been proved that the disease is transmitted to man by a certain wood tick, which receives the virus from infected wild rodents, especially gophers and jack-rabbits. Ricketts was the first to transmit the disease from man to guinea-pigs and monkeys by injecting the blood drawn from spotted-fever patients, and having fed normal ticks on the infected guinea-pigs, to reproduce the disease again to normal guinea-pigs by allowing the infected ticks to feed on the animals. Previous attempts by Ricketts and Gomez to enhance the potency of a serum which would protect against the virus by repeatedly injecting the immune animal with virus having been unsuccessful, NOGUCHI (*Jour. Exper. Med.*, 1923, **37**, 383) conducted a series of experiments with the idea of producing "an immune serum of at least sufficient potency to be useful when the time of infection is definitely known and the injection of serum can be made soon after the inoculation of the virus, as when a laboratory worker is bitten by an infected tick or accidently inoculates himself with an instrument charged with infected material." By inoculating the serum of rabbits, immunized by an attack of the disease plus a reinforcing infection two weeks after recovery, into guinea-pigs after the injection of the virus and at the end of twenty-four, forty-eight, seventy-two and ninety-six hours, and five, six and seven days, it was found that the early administration of such an immune serum will prevent the virus from multiplying and causing

fatal infection. When the immune rabbit serum was given to the infected guinea-pigs within the period of incubation the suppression of the infection was insured, but after the onset of symptoms the serum exerted no beneficial effect. In view of the high titer of the immune rabbit serum and of the comparative susceptibility of man and the guinea-pig, the author recommends that 0.2 cc of the serum per kilo of body weight be injected immediately after the inoculation, preferably intravenously. For the average adult about 16 cc should be given.

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**Incidence of Hemolytic Streptococci in Normal Preputial Secretions in Men.**—Pilot and Brams (*Jour. Infect. Dis.*, 1923, **32**, 172) were able to isolate and identify hemolytic streptococci from the preputial sac of 9 out of 100 normal men from twenty to forty years of age who did not exhibit genital lesions. The individuals examined were from the poorer walks of life. The streptococci always occurred in small numbers and presented the characteristics of Streptococcus pyogenes. They were somewhat less pathogenic than streptococci isolated from tonsils and adenoids. Staphylococci were encountered in large numbers constantly in the cultures from the preputial secretions. The coccal flora of the preputial sac resembled that of most moist hairy regions of the body. The authors state that "In analyzing the data now available as to the distribution of hemolytic streptococci in normal persons, we must recognize that their occurrence in regions other than mouth is unusual, and that the most constant habitat is the oro- and nasopharynx, particularly in the crypts of the tonsils."

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**Intestinal Antisepsis: Effect of Antiseptics on a Type of Experimental Toxemia.**—Having previously demonstrated that the toxins, whose absorption is responsible for the toxemia following acute intestinal obstruction, are produced chiefly by the activity of the proteolytic group of intestinal bacteria, Dragstedt, Dragstedt and Nisbet (*Jour. Lab. and Clin. Med.*, 1922, **8**, 190) conducted over 100 experiments on dogs to ascertain the effect of various antiseptics on the bacterial flora of closed intestinal segments. These isolated segments of the upper jejunum were made by dividing the bowel in two places from 8 to 12 inches apart and then reuniting the proximal and distal intestine around the isolated segment, care being taken not to injure the blood supply to the segment. In most cases the intestinal segment was first thoroughly washed with water and then with the antiseptic to be tested. The category of antiseptics employed included ether, alcohol (70 per cent), lysol (2 per cent), silver nitrate (5 per cent), thymol (10 per cent), Dakin's solution, betanaphthol, mercuric chloride and saturated solutions of zinc chloride, salol or quinine sulphate. Approximately, 90 per cent of the animals died within a week following the operation and at autopsy there was found usually a marked distention and perforation of the perforated loop with general peritonitis. Although these materials were applied directly to the segment of intestine in their given strength undiluted or mixed with gastric or intestinal content, it was not possible to secure a sterilization or to prevent the intestinal toxemia as produced under the conditions of these experiments.

## HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

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**A Study of Malaria Prevalence and Some of the Factors Affecting it in the Sikeston Area of Southeast Missouri.**—ZIEGLER and MAXCY (*Pub. Health Repts.*, 1923, **38**, 237), in discussing their survey, point out that malaria is still an important problem, though the incidence is on the decline. In one locality 15 per cent of persons had malaria each year. The tenant farmer is held partly responsible, due to the shifting nature of this population and the consequent lack of interest in improvement. The last paragraph of the paper contains the following useful suggestions: At present, efforts to reduce malaria morbidity should center chiefly about the individual home and the school. The people must be educated to the role of the mosquito in transmission and its life history and habits; also how properly to screen their homes and the value of keeping them tightly screened, and how to "hand-catch" the adults that rest in bedrooms by day. Some provision for more adequate medical attention must be made. Quinine must be popularized in place of the inadequate "chill tonics" and the "standard treatment" should be introduced in order to diminish the percentage of "carriers."

**Tuberculosis: A Chapter of a Forthcoming Report of the Committee on Municipal Health Department Practice of the American Public Health Association.**—WINSLOW and BAKER (*Am. Rev. Tuberc.*, 1922, **6**, 960) state that perhaps the most remarkable feature brought out by this study is the fact that while efficient antituberculosis work is carried on in nearly all cities by some agency, the municipal health department plays, in most cases, a very small part in this important field of public health. In only 46 out of 83 cities are there municipal tuberculosis clinics; in only 45 are there tuberculosis nurses; in only 29 has the health department any specific appropriation definitely ear-marked for tuberculosis work; and in only 13 is there a separate bureau of tuberculosis and in only 6 a full-time director. The budgetary allotment for 23 cities having a definite allowance is 4.9 cents *per capita*. The authors believe that every health department in a city of 100,000 population should include on its staff an expert, specifically charged with the supervision and coördination of the campaign against the most deadly of preventable diseases, for whatever aid may be offered by private agencies the strategy of a widely conceived antituberculosis movement can be fully executed only by

the official health authorities of the community. Sixty-six of the 83 cities studied have a law requiring the reporting and registration of tuberculosis. The enforcement of the law is far from complete, however, since of 69 cities for which data was collected, 40 show less than 2 cases reported per annual death and only 8 show more than 3 cases per annual death. Four or 5 cases per annual death would be a reasonable standard. Practically all cities have an antispitting law, but only 13 report arrests. Thirty-four cities have a law for the isolation of the wilfully careless consumptive, but only 8 report action taken under this statute. In 37 of the 77 cities where data are available, municipal health departments conduct tuberculosis clinics, and in 9 cities clinic provisions are secured by other municipal agencies, 5 of the first and 2 of the second group having private clinics in addition. Eleven cities have state or county supervised clinics, in some instances coöperating with private or municipal agencies. Private organizations alone provide 20 cities with clinic facilities. The number of sessions per clinic averages 4.7 per week, and 2.7 per week per 100,000 population. Evening sessions are held in 12 cities. The average number of physicians engaged in tuberculosis clinic service is 1.5 per 100,000 population. From the statements available, it appears that approximately 10 per cent of the total number of physicians render whole-time service. In several cities the medical service is voluntary. Until a paid medical service is provided the best results will not be obtained from the clinic as a factor in the antituberculosis movement. The average number of nurses engaged in tuberculosis clinic work is 2.3 per 100,000 population. In those instances where specialized tuberculosis work is carried out by the Health Department, a slightly higher rate, or 2.7 per 100,000, is found. The ratio of both physicians and nurses to population is far too low for adequate results. The average number of annual visits to tuberculosis clinics per 100,000 population is 2228. This somewhat surprisingly high figure is due to the work accomplished in the larger cities. For 41 cities below 250,000 population the average visits per year per 100,000 population amount only to 1432. The average number of visits per patient per year, on the other hand, is only 2.8, and the average number per new patients is 6.9. An average of less than 3 visits per patient per year seems to reflect a distinct lack of efficiency in holding the patient. The average cost of clinic service per visit and per patient is $1.84 and $4.76 respectively. Institutional facilities for the treatment of tuberculosis are made available for city cases through municipal departments of health, hospitals, the state, the county or private organizations. The average number of institutional beds available for city tuberculosis patients per 100,000 population is 59.6, or 1 bed for approximately every 2 annual deaths, as against the usual standard of 1 bed per every annual tuberculosis death. The percentage of these beds occupied averaged 78. The admission rate to hospitals and sanatoria is 64 per 100,000 population, or 52.9 per 100 annual deaths. As is well known, the condition of the patient on admission to the hospital or sanatorium is, in a large measure, advanced. In the present study about 60 per cent were in the advanced stage and about 17 per cent in the incipient. The average length of stay by the patient was 6.2 months, a rather good figure; and the cost per patient per day averages $2.25. Sixty-

four cities report active follow-up work of discharged arrested cases and 50 cities claim to carry on this work until the termination of the case. The visiting nurse and the public health nurse play a most important part in this work. Sixteen cities out of 28 state that the follow-up work is done by the local visiting nurse or public health nurse association. Various methods for vocational rehabilitation of arrested cases are provided by 26 cities. Over one-half of the cities carry on special antituberculosis activities and treatment measures, as is illustrated by the fact that 30 cities have open-air schools or classrooms and 25 have camps of various types. In general, then, it may be concluded that antituberculosis work in the large cities of the United States shows encouraging progress along the lines of dispensary and sanatorium treatment and of public health nursing, but a much more extensive department is needed. The municipal health department has, however, been notably backward in taking the position of leadership which properly belongs to it. The larger cities in general show much more efficient machinery for dealing with this problem than the smaller ones. The appointment of a competent director of antituberculosis work under the municipal department of health would be a most important step in coördinating the activities of existing agencies and filling gaps, in ensuring the provision of adequate dispensary and nursing and sanatorium service, and in securing the treatment of a larger proportion of patients in the curable stages of the disease.

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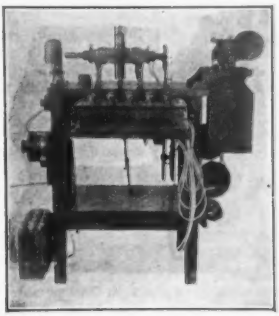

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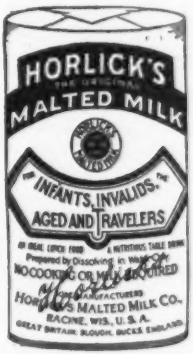
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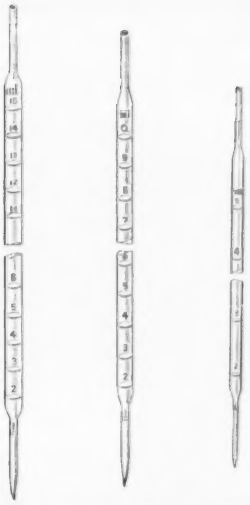

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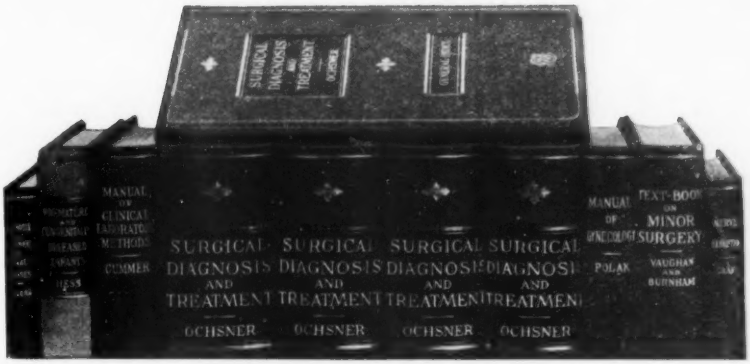


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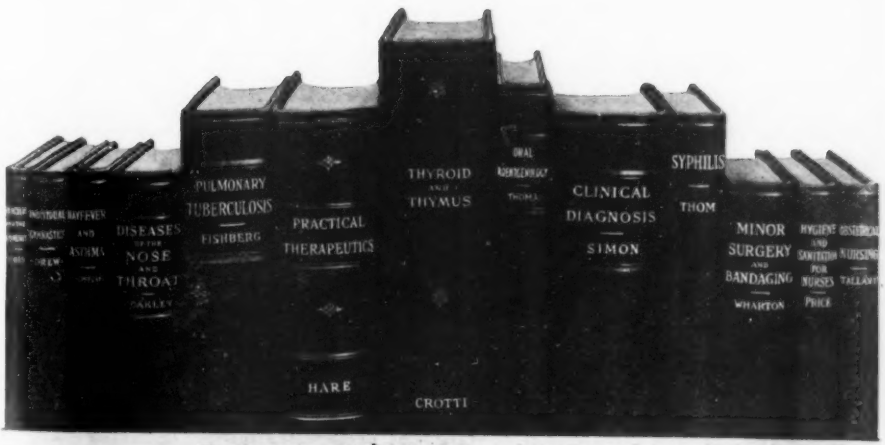


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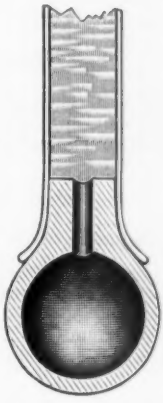

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